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Table of Contents

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Mathison Memorial Lecture.[1]

OXYGEN LACK: ITS CAUSES, EFFECTS AND RELIEF.

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WITH much diffidence I pay tribute to Mathison and thank the University of Melbourne for the very great honour of being associated with the perpetuation of his memory. By Mathison's death Australia lost a most gallant soldier and the greater world of science an investigator of proved brilliance. In 1909 and 1910, when I was a mere schoolboy, he carried out a series of critical researches analysing in detail the effects of asphyxia and studying the respective parts played by carbon dioxide retention and oxygen lack.[1][2] Hence my choice of subject for this lecture.

It was a major tragedy that not until Mathison had been killed did the authorities realize that urgent practical problems arising out of aviation, chemical warfare, wound shock and food shortage could be dealt with only by the methods of trained physiologists. Such people were not numerous. The effects, prevention and treatment of oxygen lack in aviation and chemical warfare became so important that a large number of special workers was needed. Douglas, J. G. Priestley and Bazett were recalled from duty as ordinary medical officers. The services of J. S. Haldane, Barcroft, Meakins, Shaw Dunn, Leonard Hill, Flack, Dreyer and many others were also availed of. Their work is ably summarized in the "Official History of the War, Medical Services, Diseases of the War", Volume II (an excellent textbook of applied physiology). Had Mathison sur-

[1] Delivered at the University of Melbourne, August 8, 1938.

vived it is certain that his name would have figured prominently in that story. I trust and believe that the lessons of the last war have not been lost, and, should we be faced with another national catastrophe, the services of highly trained scientists and specialists will from the very beginning be utilized in ways in which they can be most useful.

Barcroft[3] classified oxygen lack into three types: (i) anoxic, where there is inadequate oxygenation of the blood passing through the pulmonary capillaries; (ii) anæmic, where the oxygen carrying power of the blood is deficient, either from insufficiency of hæmoglobin or its combination with some other gas; (iii) stagnant, where the blood flow is insufficient to provide adequate oxygen supplies to the tissues, locally or generally.

Causes of Oxygen Lack.

Time does not permit discussion of all the numerous causes of these various types. Most of them will be already familiar. There are, however, two which I should like to speak of in some detail.

Rapid Shallow Breathing.

The first of these may perhaps best be discussed historically. During 1917 and 1918, Haldane, Meakins and Priestley,[4] at the Fifteenth Canadian General Hospital, Taplow, Buckinghamshire, set out to investigate the chronic symptoms which sometimes followed poisoning by lung irritants. Other workers had observed slight polycythæmia, slight reduction in the alveolar carbon dioxide percentage of the lungs, and a slight diminution in the carbon dioxide combining power of the blood. A similar group of phenomena had previously been observed in mountain sickness and could be attributed to a mild degree of oxygen lack. The patients became short of breath on the slightest exertion and also suffered from palpitation, precordial pain, vertigo, fatigue and orthopnœa. When the exercise was carried out in an atmosphere enriched with oxygen from a small mine rescue apparatus the subjective relief was obvious. This confirmed the hypothesis that an essential factor was a mild degree of oxygen lack which naturally would be increased during exercise, when the oxygen usage was greater. At first it was considered that the oxygen lack might be due to a residual thickening of the cell layer or layers between the air of the lung alveoli and the pulmonary capillaries. Apart from lack of histological confirmation, this idea was negatived by the fact that similar phenomena occurred in patients who had never been "gassed". These were certain cases of "effort syndrome" and others classed as war neurasthenia and shell-shock. The most striking symptom was rapid shallow breathing. In normal individuals undertaking slight to moderate exercise the predominent factor of the increased lung ventilation is increase in depth. In these cases, however, the predominant factor was increase in rate, sometimes even to 60 or 70 per minute, as a result of such mild exercise as mere walking.

This led Haldane, Meakins and Priestley,[5] using themselves as subjects, to investigate the respiratory response to progressive lack of oxygen. They found that it occurred in three stages: (*a*) increased depth of breathing, (*b*) Cheyne-Stokes breathing, (*c*) increased rate with diminished depth.

They then proceeded[6] to study the effects of restricted depth of breathing, and found that rapid shallow breathing produced symptoms indicative of oxygen lack. They argued from the earlier observations of Keith[7] that the expansion of the lungs was not equal throughout and therefore the ventilation of the lungs was not the same in all parts. In rapid shallow breathing the inequalities of ventilation would be exaggerated. From the shape of the oxyhæmoglobin dissociation curve of the blood, which attains a plateau of almost full oxygen saturation at a pressure of oxygen a little beyond 100 millimetres of mercury, it is obvious that a well-ventilated part of the lung cannot compensate for badly ventilated parts. So the oxygen saturation of the mixed arterial blood will be less than might be inferred from the mean alveolar oxygen pressure. The net result is that rapid shallow breathing is a cause of oxygen lack. At the time of these original experiments the technique of arterial puncture had not been introduced into Great Britain, so direct proof of the existence of oxygen lack was still wanting. In 1920,[8] however, the experiment was repeated, Haldane being the subject, with the results shown in Table I. Thus it can

Table I.

Respiratory Rate per Minute.	Respiratory Minute Volume in Litres.	Mean Tidal Air in Cubic Centimetres.	Arterial Oxygen Saturation *per centum*.	Expired Carbon Dioxide in Cubic Centimetres per Minute.
17·5	10·3	586	94·3	359
35·0	13·5	385	93·6	388
48·0	15·9	315	91·7	397

be seen that in spite of a considerable increase in respiratory minute volume, restriction of depth of breathing caused a fall in arterial oxygen saturation. This was a splendid vindication of the argument and proved beyond all question that rapid shallow breathing could be a cause of oxygen lack. From their previous work we have seen that it is a result of oxygen lack. Hence a vicious circle may be set up in a variety of clinical respiratory disorders. Doubtless this was the underlying feature in the cases mentioned above.

Excessive Elimination of Carbon Dioxide.

We shall now consider a second cause of oxygen lack. It may be noted that in the experiment on Haldane previously cited (Table I) there was an increased output of carbon dioxide. This we can regard as due to increased respiratory minute volume brought about by oxygen lack, which as we now know acts upon chemical receptors in the

carotid body, sending afferent impulses to the respiratory centre. (Heymans *et alii*,[9] whose work has been recently confirmed by Comroe and Schmidt.[10]) In the case of carbon dioxide there is no plateau on the dissociation curve, so that well-ventilated parts of the lungs can fully compensate for badly ventilated parts. The result of this excessive carbon dioxide elimination is a reduced partial pressure of carbon dioxide in the blood ("gaseous alkalosis"), and consequently, as can be seen from the classical work of Bohr *et alii*,[11] an increased stability of oxyhæmoglobin. Thus although a sufficiency of oxygen may be present in the blood, it is less readily available to the tissues. This condition may be termed "secondary oxygen lack"; it occurs in any case of oxygen lack in which there is no obstacle to the removal of carbon dioxide. Such a condition occurs in mountain sickness and in miners overcome by "black damp". It also occurs in carbon monoxide poisoning. I have seen severe cases in which the gaseous alkalosis was sufficient to cause muscular rigidity approximating to tetany. In such cases the relief following administration of carbon dioxide and oxygen was quite dramatic.

This factor of secondary oxygen lack is important clinically. In cases in which œdematous or inflammatory thickening is limited mainly to the alveolar walls, carbon dioxide, by virtue of its great solubility and diffusibility, is not retained; but oxygen lack occurs. On the other hand, where the lesion obstructs the finer bronchioles there is insufficient surface area for diffusion and oxygen lack is associated with carbon dioxide retention. Examples of these different conditions are lobar pneumonia as compared with bronchopneumonia, pulmonary œdema with acute bronchitis, and phosgene poisoning with mustard-gas poisoning. In extreme cases the difference in symptoms is obvious. With secondary oxygen lack there may be some muscular rigidity. There are slight if any subjective sensations of dyspnœa (apart from pleural pain) and the cyanosis is of the pallid leaden type. On the other hand, with carbon dioxide retention dyspnœa may be severe and the cyanosis is of the plethoric plum-coloured type. With less severe cases the differences are less obvious and call for nice judgement on the part of the clinician as to whether or not the addition of carbon dioxide to the oxygen therapy be desirable. J. S. Haldane once told me (I have searched in vain for the original published data) that Paul Bert was very much puzzled by the fact that with animals confined in closed vessels the lethal point of oxygen deficiency was much lower when carbon dioxide was allowed to accumulate than when it was absorbed by alkali. There are other well-known cases in which the effects of secondary oxygen lack are apparent, such as in carbon monoxide poisoning, following explosions in mines (afterdamp) or exposure to the exhaust fumes of internal combustion engines. In these and other cases the carbon monoxide is associated with a high percentage of carbon dioxide, and unless the concentration of the former gas be overwhelming the presence of the latter enables the victim to reach fresh air where carbon dioxide is no longer present in significant amount and the patient immediately collapses. Haldane once told me that a time-honoured method among miners for treating such patients was to cut a small sod of turf and place the victim's face in the hole. This would cause partial rebreathing and consequently an increase in the carbon dioxide content of the blood. The modern method of resuscitation is by administration of carbon dioxide with or without oxygen.

I have dwelt somewhat at length on the subject of secondary oxygen lack because I am convinced that its importance may not always be fully realized. Ample theoretical justification as well as practical experience fully supports the case for the therapeutic exhibition of carbon dioxide. It is satisfactory to note that its use is becoming more frequent.

The Effects of Oxygen Lack.

The effects of oxygen lack are sufficiently well known to need little exposition. The most important point is that if it is allowed to persist it causes damage to important organs—damage which is repaired slowly, if at all. The extent of that damage will depend upon the severity and duration of the oxygen lack. Barcroft[3] misquotes (or "sloganizes") Haldane in the statement that "Anoxæmia not only stops the machine but wrecks the machinery". It is unfortunate that his misquotation, the entire negation of Haldane's philosophical standpoint,[12][13] has been perpetuated in one of the most widely read English text-books of physiology.[14] What Haldane[15] actually said was this, and it might well be inscribed in letters of gold on the walls of every department of physiology:

> I need hardly remind you that the place of oxygen in the life of a warm-blooded animal is altogether peculiar, since the body has practically no storage capacity for oxygen, but depends from moment to moment for its supply from the air. If we cut off this supply loss of consciousness is only a matter of seconds, and death of minutes. As Paul Bert was the first to point out, the immediate cause of death is practically always anoxæmia. But death is not the mere stoppage of a machine; it is also the total ruin of the supposed machinery. Similarly—and this is a lesson which I wish to emphasize as strongly as I can—partial anoxæmia means not a mere slowing down of life, but progressive and perhaps irreparable damage to living structure.

To add anything further would be not only superfluous but impertinent.

The clinical signs of oxygen lack need only brief mention. They are discussed by Campbell and Poulton,[16] also by Meakins and Davies.[17] The most important are as follows.

1. *Cyanosis.*—Cyanosis is of doubtful value as a sign of oxygen lack. It is apparent in normal persons (for example, in mountain sickness) only when the arterial oxygen saturation falls to 85% or 80%.[18] In anæmic people it may be absent. In plethoric people it may be unimportant. When there

is much skin pigmentation or in a bad light it may be unobservable.[19] It is best detected by its disappearance when oxygen is effectively administered.

2. *Respiratory Rate.*—Any increase in the rate of respiration above 30 per minute demands serious consideration for reasons which have already been discussed.

3. *Orthopnœa and Cheyne-Stokes Breathing.*—Orthopnœa and Cheyne-Stokes breathing are definite indications of oxygen lack.

4. *Restlessness and Mild Delirium.*—When associated with pulmonary lesions restlessness and delirium must be regarded seriously.

5. *Increased Pulse Rate.*—An increase in the pulse rate invariably accompanies oxygen lack except in the later stages when, as Mathison[20] showed, heart block may occur.

The combination of two or more of these signs or symptoms demands serious consideration. Dyspnœa, unless due to pleural pain, is a sign of carbon dioxide retention rather than of oxygen lack.

Prevention and Treatment.

We shall now consider the prevention and relief of oxygen lack; but first it seems desirable to deal with various fallacies and misrepresentations regarding oxygen administration. It is unfortunate that so valuable and life-saving a remedy should have fallen into a degree of disrepute. When we read in the newspapers that oxygen has been administered to a royal personage we immediately shake the moth-balls from our ceremonial garments. The public generally have been apt to regard oxygen administration as but an expensive and futile preliminary to the services of the undertaker. The reason for this is doubtless inefficiency or undue delay in its administration. Very few medical practitioners approach the matter of oxygen administration quantitatively. Some of the methods still in use even in teaching hospitals might be likened to sprinkling a dose of medicine on the bedclothes instead of giving it by mouth. For efficient oxygen administration it is necessary to have a clear conception of the object to be attained. In "anoxic" oxygen lack due to pulmonary causes it is necessary to raise the partial pressure of oxygen in the alveolar air sufficiently to overcome the resistance of the pulmonary lesion and to restore the normal oxygen saturation of the arterial blood. In most cases this object can be attained by raising the oxygen percentage in the inspired air to 35 or 40. In "anæmic" oxygen lack, such as severe carbon monoxide poisoning, it is necessary to raise the inspired oxygen percentage to as high as possible (90 to 95) in order to increase the amount of oxygen in simple physical solution in the blood plasma.

But before considering effective means it is necessary to deal with certain stupidities perpetrated in the name of oxygen administration and with certain excuses for withholding oxygen when intelligent appreciation of the situation would clearly indicate its necessity. These may be itemized as follows.

1. *Inadequate Rate of Flow.*—Not infrequently when visiting a hospital one sees oxygen bubbling at easily countable rates through a wash-bottle to a patient. Now the maximum rate at which I can count bubbles is about five per second—one appreciates them in groups like demisemiquavers. With a tube of a bore of about six millimetres this corresponds to a rate of oxygen flow of about a quarter of a litre per minute. But about half of this oxygen is entirely wasted—it is blown away during the expiratory phase of breathing and never enters the patient's lungs. Therefore one can say that the effective rate of oxygen flow is about one-eighth of a litre per minute. Assuming that the patient is an adult suffering from pneumonia, the respiratory minute volume would be of the order of ten litres per minute. The addition of an eighth of a litre of oxygen would increase the inspired oxygen percentage from the normal 21 to slightly above 22. Veritably a drop in the bucket.

2. *Intermittent Administration.*—As has already been seen, the body has no appreciable storage capacity for oxygen. Administration for five minutes every hour is, as I tell my students, like having a drowning man on the end of a line and hauling him up to the surface for five seconds each minute.

3. *Freak Methods (in Particular Oxygen Injections).*—Campbell and Poulton,[16] discussing a case in which 500 cubic centimetres of oxygen had been injected subcutaneously and some remained unabsorbed after forty-eight hours, point out that the rate of absorption could not have exceeded 0·18 cubic centimetre per minute, whereas a normal man at rest requires about 250 cubic centimetres per minute. Moreover, if one considers the general circulation, it can be seen that oxygen injected over the widest practicable area subcutaneously or intraperitoneally will serve to oxygenate only an insignificant fraction of the mixed venous blood. The analogy I use with my students is that of pouring buckets of water into a river with the object of raising the water level many miles upstream. It was shown quite clearly by Rabinovich (now Robson) and myself[22] that experimental oxygen lack which could readily be overcome by oxygen inhalation was not appreciably affected by oxygen injections. Detailed figures for these experiments are given in Table II. The animals were anæsthetized with "Luminal Sodium", which probably accounts for the somewhat low initial arterial oxygen saturations.

It was interesting to read, in a previous Mathison Memorial Lecture, that Professor Osborne[23] had carried out some experiments with intravenous injections of oxygen, but was unable to advise the use of this procedure in oxygen lack in man. The reasons were not given in the published lecture; but I am in emphatic agreement. On a number of occasions, using anæsthetized dogs rendered

Table II.

The Effects of the Injection of Oxygen Compared with the Effects of Breathing Oxygen.

Number of Dog.	Weight of Dog in Kilograms.	Percentage Saturation of Oxygen in Arterial Blood.				
		Normal.	Anoxæmic.	After Subcutaneous Injection of Oxygen.[1]	After Intraperitoneal Injection of Oxygen.[1]	After Breathing Oxygen.
I	14·0	92·0	85·0	87·0 (150)	86·0 (1,000)	—
II	23·0	89·0	81·0	81·0 (150)	83·0 (1,000)	94·0
III	13·0	88·0	78·0	87·0 (200)	—	97·0
V	19·0	87·0	80·0	85·0 (350)	82·0 (500)	95·0
VI	17·0	86·0	56·0	43·5 (300)	—	94·5
VIII	15·0	85·0	80·0	80·0 (1,000)	—	98·0
X	13·0	91·5	56·0	53·0 (500)	53·0 (500)	94·5

[1] The quantity of oxygen, in cubic centimetres, injected in any particular instance is shown in parentheses.

anoxæmic by multiple pulmonary emboli, Rabinovich and I found that sudden death followed intravenous injections both of oxygen and of hydrogen peroxide. The reason, I think, is that the bubbles of oxygen attain gaseous equilibrium with the dissolved nitrogen and carbon dioxide of the blood plasma and therefore remain as bubbles and produce multiple capillary air locks.

One must admit, however, that Oliver and Murphy,[24] cited by Campbell and Poulton,[16] observed a striking reduction of mortality in influenzal pneumonia following the intravenous injection of hydrogen peroxide diluted with normal saline solution and rendered alkaline by *liquor ammoniæ*.

4. *Unsuitable Apparatus.* — Any appreciable respiratory resistance or undue increase of dead space is intolerable to a patient suffering from oxygen lack. It is essential to ensure that the apparatus is working properly and the oxygen flowing freely before applying the mask to the patient. But in this connexion it must be pointed out that even the most skilfully devised apparatus may fail owing to faulty manufacture, misuse or insufficient attention to instructions.

5. *Delay in Administration.*—The administration of oxygen is sometimes delayed until the patient is *in extremis*. Oxygen lack must be forestalled or at least remedied at its first appearance. Thus the delirium and cyanosis in the early stages of pneumonia can readily be controlled by efficient oxygen administration; but if it is left unrelieved more permanent damage results, and delirium, coma and circulatory failure remain, despite oxygen administration. Moreover, restlessness and delirium cause an increased demand for oxygen, so that a vicious circle is set up.

6. *Toxicity of Oxygen.*—It is true that mice kept in pure oxygen for about three days develop signs of pulmonary irritation, also that dogs exposed to oxygen at a pressure of about three atmospheres show symptoms similar to those of strychnine poisoning; but an overdose of any potent therapeutic agent will cause untoward effects. There is absolutely no evidence that therapeutic concentrations of oxygen produce any ill effects. To withhold oxygen from a patient suffering from oxygen lack is as illogical as to deny food to a starving man on the grounds that others have suffered ill effects from gluttony.

7. *Expense and Clumsiness of the Apparatus.*—The apparatus is no longer expensive and cumbersome. In the principal cities of Australia the group of firms who manufacture the bulk of our oxygen provides oxygen service at a cost which, for so life-saving a remedy, cannot be regarded as excessive. For hire of apparatus the charge in Sydney is 21s. for the first week or part thereof, and 15s. for subsequent weeks. Oxygen costs 8s. 6d. per 100 cubic feet in cylinders holding 100 cubic feet each. In metropolitan areas these costs include transport and technical assistance. It remains for the clinician only to ensure that the patient's needs are adequately supplied.

The General Principles of the Administration of Oxygen.

If any of my audience have been disappointed with oxygen therapy in the past, I trust that the reasons are now apparent—inadequate, inefficient or tardy administration—and I beg that they will try again, using one of the more satisfactory methods now available. Here I must give a warning. In the literature circulated by the local oxygen firms my name appears in connexion with certain apparatus which I consider satisfactory. I would here disclaim any financial interest. I have, if I may say so modestly, strictly followed the example of my revered friend and teacher, the late J. S. Haldane, in refusing to patent any appliance which might be used for the relief of human suffering. Frequently in the advertising pages of The Medical Journal of Australia and in the same literature there is depicted and described an oxygen tent which I strenuously refuse to endorse. It is made of porous fabric, and some time ago Dr. A. D. Gillies, then a student in my department, found that it required ten litres of oxygen per minute to maintain an atmosphere containing 40% of oxygen. At this rate of oxygen flow a 100 cubic foot cylinder would last for less than five hours. Yet the manufacturers claim it as the most efficient method of administering oxygen or "Carbogen". With a really efficient apparatus the same result can be attained with two litres of oxygen per minute, so that a 100 cubic foot cylinder lasts for nearly twenty-four hours. Moreover, atmospheric air contains approximately 20% of oxygen, and if the added 20% be supplied

by a mixture consisting of 95% of oxygen and 5% of carbon dioxide it can be seen that the resultant concentration of carbon dioxide will be only 1%. Even allowing a certain amount of rebreathed carbon dioxide from the patient's expired air, the percentage of carbon dioxide will be therapeutically almost negligible. I have heard that this outfit is sometimes sent to patients in the country. We have seen that when it is used a cylinder containing 100 cubic feet of oxygen suffices for a period of less than five hours of effective administration. Therefore we may say that in such circumstances, even if it arrives in time, it can only afford the unfortunate patient a lucid interval sufficient to settle his worldly and ghostly affairs.

We have already seen that effective administration of oxygen involves an adequate increase of oxygen percentage in the air in the lung alveoli. Normally, in round numbers, atmospheric air contains 20% of oxygen and alveolar air 14%—a difference of about 6%. To raise the oxygen percentage in the alveolar air in a quantitative manner it is necessary to know two things: first, the respiratory minute volume, and secondly, the effective rate of oxygen flow (*E.O.F.*). This second factor needs explanation. Supposing we administer oxygen to a patient through a nasopharyngeal catheter at the rate of two litres per minute. During the expiratory phase the oxygen flowing is blown away with the expired air and never enters the lungs at all. Since the two phases of respiration are approximately equal, we can say that about half of the oxygen is wasted and in this case the effective rate is one litre per minute. So that by effective rate we mean the amount of oxygen added to the inspired air. Now suppose that the respiratory minute volume (*R.M.V.*) of our patient be five litres per minute. This would consist of four litres of room air together with the one litre of effectively added oxygen. Since room air contains approximately one-fifth of oxygen the total inspired oxygen per minute is four-fifths of a litre *plus* one litre, which, the total respiratory minute volume being five litres, corresponds to an inspired oxygen percentage of 36 and an alveolar oxygen percentage of about 30. In a similar manner we can calculate the alveolar oxygen percentage for any given effective rate of oxygen flow and any given respiratory minute volume. Results of such calculations are given in Table III.

It may be noted that for any given ratio of effective oxygen flow to respiratory minute volume the alveolar oxygen percentage is constant. This enables the table to be simplified. This has been done in Table IV. Also one may construct a D'Ocagne nomogram relating the three factors. This has been done in Figure I. Here one stretches a thread or lays a straightedge through the two points on the *R.M.V.* and *E.O.F.* scales and notes the point at which it cuts the inspired oxygen scale. The reading here, less six, gives the theoretical alveolar oxygen percentage. Conversely, by drawing a line through the *R.M.V.* and the desired inspired oxygen percentage one can read off the necessary *E.O.F.*

From this nomogram we can also calculate the efficiency of any given method. Thus, supposing in a particular case the calculated alveolar oxygen percentage be 39, whereas by sampling and analysis we find it to be only 34, we can state that the method of administration is only 80% efficient.

$$\frac{34-14}{39-14} \times 100 = 80\%$$

In this manner the percentage efficiency of a number of methods was determined by Gilchrist and myself.[25] The results are given in Table V. This table also enables me to correct a common fallacy in current literature, namely, that the nasopharyngeal catheter is far superior to the time-

Table III.

Showing the Percentage of Oxygen in the Alveolar Air in Relation to the Rate of Effective Oxygen Flow (E.O.F.) and the Respiratory Minute Volume in Litres (R.M.V.).

Rate of Effective Oxygen Flow (*E.O.F.*) in Litres per Minute.	The Percentage of Oxygen in the Alveolar Air.											
	R.M.V. = 3	*R.M.V.* = 4	*R.M.V.* = 5	*R.M.V.* = 6	*R.M.V.* = 7	*R.M.V.* = 8	*R.M.V.* = 9	*R.M.V.* = 10	*R.M.V.* = 11	*R.M.V.* = 12	*R.M.V.* = 13	*R.M.V.* = 14
1	41	34	30	28	26	24	23	22	22	21	21	20
2	67	54	46	41	37	34	32	30	29	28	27	26
3	94	74	62	54	48	44	41	38	36	34	33	32
4	—	94	78	67	60	54	50	46	43	41	39	37
5	—	—	94	80	71	64	58	54	50	47	45	43
6	—	—	—	94	82	74	67	63	58	54	51	48
7	—	—	—	—	94	84	76	70	65	61	57	54
8	—	—	—	—	—	94	85	78	72	67	63	60
9	—	—	—	—	—	—	94	86	79	74	69	65
10	—	—	—	—	—	—	—	94	86	80	75	71
11	—	—	—	—	—	—	—	—	94	87	81	77
12	—	—	—	—	—	—	—	—	—	94	87	82

Table IV.

The Percentage of Oxygen in the Alveolar Air in Relation to the Ratio of Effective Oxygen Flow to Respiratory Minute Volume (E.O.F. : R.M.V.).

Ratio *E.O.F.* : *R.M.V.*	1:1	1:2	1:3	1:4	1:5	1:6	1:7	1:10	1:15
Percentage of oxygen in alveolar air	94	54	41	34	30	28	26	22	20

honoured glass funnel. Provided the latter is of large diameter and closely approximates the patient's face, so that the rim touches the bridge of the nose and the chin, there is little to choose between the two methods. Both are approximately 50% efficient, which is just what one would expect, because the flow of oxygen is continuous and the effective rate, as previously mentioned, is about half the actual rate of oxygen flow. Using the glass funnel in the above-described manner in a case of, say, pneumonia, with a respiratory minute volume of eight litres and an oxygen flow of four litres per minute (*E.O.F.* two litres), we can see that the percentage of oxygen in the alveolar air will be 34, and in the inspired air 40. To produce the same inspired oxygen percentage with the leaky tent mentioned above, ten litres of oxygen per minute are necessary; the glass funnel is therefore two and a half times more efficient. Now let us try four litres of oxygen by means of the nasal catheter, which many text-books recommend. From personal experience I can say that it feels as though one's nasopharynx had been invaded by a swarm of bees. Although both methods are almost equally wasteful, one can say that the glass funnel permits adequate administration, whereas with the nasopharyngeal catheter an adequate rate of oxygen flow is intolerable.

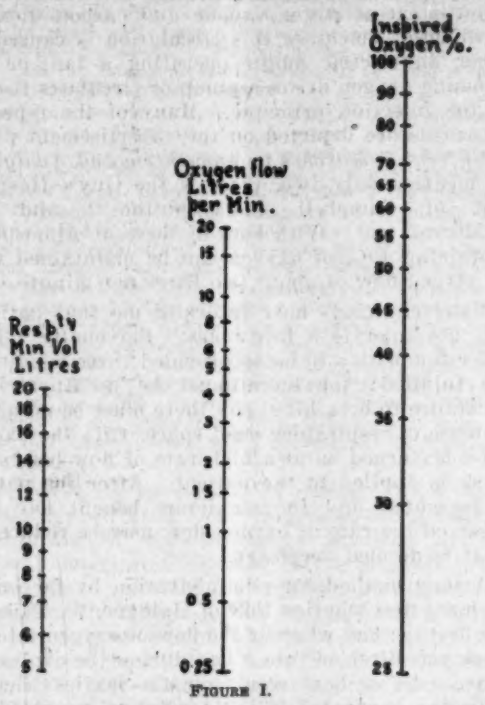

FIGURE I.

Clinical Criteria of the Efficiency of a Method.

So much for the general principles involved in oxygen administration. We may now consider how to determine whether in any particular case they have been adequately fulfilled. Ideally, I suppose, one should check the efficiency of administration by blood gas analyses. This, however, is seldom feasible, and would in any case be somewhat tiresome for the patient. There are, however, clinical criteria of effective administration which are quite satisfactory. These may be discussed in some detail.

1. *Reduction in Pulse Rate.*—The pulse rate becomes slower even in normal individuals when

TABLE V.

The Results obtained from Tests of the Efficiency of Various Methods of Oxygen Administration.

Method.	Rate of Dioxide Flow in Litres per Minute.	Respiratory Minute Volume in Litres.	Rate of Dioxide Flow to Respiratory Minute Volume.	Calculated Percentage of Alveolar Dioxide. See Table III	Observed Alveolar Dioxide Percentage	Efficiency Expressed as Percentage.	Remarks.
Tube and funnel	2·0	6·2	0·32	40	27	49	Funnel touching chin and nose.
	4·0	6·1	0·65	67	29	29	Funnel edge two inches away from tip of nose.
	8·0	6·2	1·29	94	47	41	Funnel touching nose and chin.
Nasal catheter	2·0	6·2	0·32	40	28	53	—
	4·0	6·7	0·60	62	33	40	—
	8·0	6·2	1·29	94	43	37	Great discomfort at this rate of flow.
Haldane apparatus	2·0	9·5	0·21	31	32	100 (+)	—
	3·0	8·7	0·35	42	44	100 (+)	—
	4·0	8·5	0·47	52	50	95	—
	8·0	9·0	0·89	85	69	77	—
	9·5	9·2	1·03	94	67	66	—
New outfit with forked nasal tube..	2·0	6·2	0·32	40	42	100 (+)	—
	4·0	8·8	0·45	51	50	97	—
	8·0	8·7	0·92	90	65	67	—
New outfit with mask	10·0	10·7	0·93	91	90	99	—
	9·0	8·9	1·01	94	92	97	—
	5·0	10·5	0·47	53	53	100	—
	4·0	10·5	0·37	45	44	97	—
	2·0	10·5	0·19	30	30	100	—

a high percentage of oxygen is breathed. In cases of oxygen lack the reduction is dramatic if oxygen is administered efficiently and by a method which is free from discomfort to the patient. The only exception to this fall of pulse rate is in cases in which heart block has supervened.

2. *Reduced Respiratory Rate.*—There is usually some reduction in the rate of respiration, but not usually to normal. This is probably because the increased rate may be due in part to afferent nerve impulses set up by the pulmonary lesions. In this connexion one may cite some experiments by Binger, Brow and Branch,[26] who produced oxygen lack in dogs by multiple pulmonary emboli. In the first case they produced multiple capillary emboli by the injection of a suspension of potato starch-granules. In this case oxygen administration overcame the oxygen lack; but the respiratory rate, although reduced, remained considerably above normal. In the second case they introduced sweetpea seeds, blocking large branches of the pulmonary arteries. Here the oxygen lack was abolished and the respiratory rate returned to normal when the animals inhaled oxygen-enriched air.

3. *Relief from Dyspnœa and Orthopnœa.*—Loss of dyspnœa and orthopnœa is frequently incomplete because these symptoms may be partly due to pleural friction. Indeed, the relief of oxygen lack may restore the acuity of pain sensation and the patient may feel subjectively worse unless morphine is administered.

4. *Abolition of Cyanosis.*—The disappearance of cyanosis is a most useful criterion if it occurs; but, as we have already seen, there may be an appreciable degree of oxygen lack without cyanosis, or the cyanosis may be not apparent owing to skin pigmentation or bad light.

5. *Abolition of Restlessness and Delirium.*—Restlessness and delirium vanish unless the oxygen lack has been severe or prolonged. When oxygen is administered effectively and without discomfort it is sometimes quite dramatic to see a restless semi-delirious patient after a few minutes passing into quiet restful sleep. On the other hand, return of these symptoms in a patient previously comatose may be a sign of improvement.

Brief Discussion of Particular Methods of Administration.

Time would not permit a detailed description of all the methods for oxygen administration. We have already discussed some of them. Others call for brief mention.

The ideal method of oxygen administration is by means of an oxygen or compressed-air chamber. But these are expensive to install and to maintain and demand constant expert supervision. Moreover, an oxygen chamber needs the most elaborate precautions against fire. I know of two instances when the contents of the chamber have taken fire and the patient was incinerated. Equally satisfactory for most purposes is an efficient oxygen tent, a number of types of which are on the market. The general feature of all is that the patient is partly enclosed in a tent of proofed fabric such as rubber waterproof or oiled silk. The air of the tent is circulated through an external purifying circuit which removes excess water vapour and carbon dioxide. In different machines this circulation is caused by either an electric motor operating a fan, or the incoming oxygen drives a pump or circulates the air by the injection principle. Many of the types of apparatus are depicted on the advertisement pages of *Current Researches in Anæsthesia and Analgesia.* To mention only two, there is the Guy's Hospital tent of Campbell and Poulton[16] and the Heidbrink tent. With both of these an atmosphere containing 40% of oxygen can be maintained with an oxygen flow of about two litres per minute.

Many clinicians have informed me that patients will not tolerate a face mask. Personally I have never found this to be so provided three conditions are fulfilled: (i) there must be no appreciable resistance to breathing, (ii) there must be no undue increase of respiratory dead space, (iii) the oxygen must be turned on at a high rate of flow before the mask is applied to the patient. After the patient is reassured and the maximum benefit has been observed the rate of oxygen flow may be reduced to what is deemed necessary.

Among methods of administration by face mask we must first mention that of Haldane.[27] This was the first method whereby the flow of oxygen into the mask was diverted into a bag during the expiratory phase. As we have seen from the Davies-Gilchrist table the apparatus is 100% efficient, provided the rate of oxygen flow does not exceed one-third of the respiratory minute volume. As it is seldom necessary to exceed this ratio the apparatus can still be recommended as being the simplest and for practical purposes fully efficient. It is, however, only partly quantitative, for although the flow of oxygen can be measured it gives no indication of the patient's respiratory minute volume. In an ordinary pneumonia in an adult the respiratory minute volume ranges between eight and twelve litres, and effective oxygen therapy is achieved with between two and three litres per minute. The necessary amount can be determined by observation of the clinical criteria enumerated above. This apparatus was first introduced for the treatment of acute gas poisoning during the Great War. It has been the means of saving many lives. In my opinion it is still the most satisfactory method for general use.

Although it may have been attempting to "gild the lily", Gilchrist and I[28] set out to overcome the two partial defects of the Haldane apparatus. I think we can claim success in obtaining full efficiency at all ratios of effective oxgyen flow to respiratory minute volume, and also in obtaining sufficiently quantitative information regarding the patient's respiratory minute volume. The modifications were as follows.

The two-way rubber-curtained air orifice on the Haldane mask was replaced by an expiratory one-way valve and a

helical-shaped extra-air orifice which could be completely closed or opened to any required extent. With this orifice closed and the mask fitting without leak the whole of the patient's respiratory requirements must be supplied by the oxygen flow. The bag was made of large capacity, and for the mica valve of the Haldane apparatus we substituted a Müller water valve, which serves the double function of moistening the oxygen and affording an indication of its flow. All the tubing of the apparatus it of wide bore to avoid any respiratory resistance.

The procedure is as follows. The extra-air orifice is completely closed and the oxygen turned on at a rate of about ten litres per minute. After a few seconds have been allowed for the bag to fill, the mask is closely applied to the patient's face. The rate of oxygen flow is then adjusted so as to maintain a constant mean distension of the bag. When this has been done a reading of the flow meter gives the patient's respiratory minute volume. The necessary alveolar oxygen percentage is then estimated and the oxygen flow reduced to the amount indicated by Table IV or the nomogram. The extra-air orifice is then opened to the necessary extent. If the rate of oxygen flow does not exceed one-third of the respiratory minute volume a forked nasal tube may be substituted for the mask, any nasal obstruction being first of all abolished by one of the numerous available "decongestives". From practical experience I can state that this forked nasal tube is remarkably efficient even when the patient is breathing mainly orally.

So concludes my story. I trust that I have adequately refreshed your memories by a logical presentation of the principles involved and a sufficient description of some of the methods used. I regret that I have not presented the results of any recent investigations. As an excuse for this I can only plead that during the past few years my limited research activities have been diverted elsewhere and that a department which caters for nearly 600 students of three different years and four different faculties lacks that calm and cloistral atmosphere which one finds in institutes devoted almost entirely to research.

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THE PRESENT POSITION OF SURGERY OF THE THYREOID.[1]

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IN the rush and bustle of a busy life it is well now and then to cry a halt for a brief period to take stock of one's position and note what progress, if any, has been made. The occasion of this lecture has given me an excuse for such a review of goitre, in the surgery of which I have long been engaged.

My interest in the thyreoid gland was first aroused as a student in 1904, when most of my vacation periods were devoted to experimental work in the department of physiology. As a resident at the Royal Prince Alfred Hospital I thought I had a unique experience in having some fifteen patients with toxic goitre in the medical wards during seven months, and some two or three operated on when I changed over to the surgical side. In those days very few surgeons would operate on these patients, as it was invariably a strenuous adventure and the mortality rate was about 20%; but even in the medical wards the mortality was around 20%, and in no case was a cure effected. The reason for this (as I know now) was that only patients in advanced stages were ever admitted to hospital, and surgery was attempted only as a last resort, in the hope that the patient might be tided over a critical period and rendered capable of receiving further medical treatment.

[1] Lecture delivered at the opening of the Post-Graduate School, the Prince Henry Hospital, Sydney, on April 5, 1938.

In non-toxic cases and those in which adenomata were present the advice then given was "as long as it is doing you no harm leave it alone and put up with the disfigurement", advice which, I regret to say, is even being given on occasions today.

During my sojourn overseas in 1908-1909, I visited Vienna and saw the Kochers, father and son, at work; and although impressed by their writings, I was somewhat disappointed to see patients strapped to the table screaming with fright and the pain of inadequate local anæsthesia until reduced to quiescence from loss of blood. Removal of adenomata, ligation of the superior thyreoid arteries and hemithyreoidectomy were the operations then in vogue; and there is no doubt that "Old Man" Kocher stimulated a world-wide interest in the operative treatment of goitre.

In my early days opportunities for surgery were few and far between, as toxic goitre in all its forms was considered to be a medical disorder, and very few people with non-toxic adenoma could be prevailed upon to submit to operation. The really great advance came with the reintroduction of iodine therapy and the realization of careful preoperative treatment as a necessity for success. Local anæsthesia was still the vogue; but intratracheal anæsthesia and later the development of gas anæsthesia were introductions of great moment to thyreoid surgery.

Following Kocher, I think Crile, Charles Mayo and Halstead did more to pave the way to safer surgery in goitre than anyone else; and then Dunhill, in Australia, made us realize that in the majority of cases medical treatment of toxic goitre should be merely the preamble to operation. In 1907 he recorded his first experience of subtotal thyreoidectomy, and ever since he has persistently advocated this procedure.

There seems to be no doubt that disorders of the thyreoid gland are becoming increasingly prevalent as compared with their incidence even fifteen years ago. Dunhill has also noticed this in England. As the thyreoid is the energizing gland of the body, one wonders if it is not the added strain and stress of modern life, causing inordinate demands on its activity, that are the big factors in the increase of its disorders, especially of toxic goitre, although other factors still play their part.

Adenoma of the Thyreoid.

I shall first consider the so-called "simple" adenoma, with which I include colloid cysts. The years have shown that the qualification "simple" is one which should be discarded, as I advocated twenty years ago. Call it non-toxic or toxic, as the case may be, if you wish to qualify the fact that an adenoma is obvious upon examination; for, as time goes on, an adenoma develops in a number of ways, and is always a potential source of grave danger to the patient.

Toxicity.—As regards toxicity, there are two dangerous age periods: one approximately from thirty to thirty-five years, when a relatively acute toxic condition may supervene, and the other from forty-five to fifty, when a low-grade toxicity, often of many years' standing, results in a breakdown of the cardiac mechanism. It has been estimated that 20% of adenomatous goitres become toxic; but if it were possible to obtain true figures I believe the percentage would be higher.

Hæmorrhage.—Apart from toxicity, other troubles may supervene, the most alarming to the patient being hæmorrhage, usually into a colloid cyst, as it causes a sudden painful increase in the swelling and frequently results in respiratory embarrassment.

Mechanical Effects.—Multiple adenomata or colloid cysts are very common and often grow to a large size, causing gross disfigurement, or by intrathoracic growth give rise to pressure effects on both circulation and respiration.

Myxœdema.—Occasionally myxœdema may occur, due to atrophy of the gland tissue proper from pressure of the adenomatous mass.

"Scabbard" Trachea.—One of the great dangers from the operative viewpoint, when large adenomata are present, is the production of a "scabbard" trachea or a gross dislocation of the trachea with absorption or weakening of the cartilaginous rings. This may lead after operation to tracheal collapse, while the patient is recovering from the anæsthesia or after a spasm of coughing. It usually occurs after a violent expiratory effort, and when inspiration commences the walls of the trachea are sucked together. Strenuous efforts are made to inspire; but they increase the difficulty; cyanosis sets in rapidly, and unless an intratracheal catheter can be introduced or immediate tracheotomy performed, death occurs within a few minutes. It is a terrifying condition to witness in a semi-conscious patient and is very difficult to relieve unless adequate assistance is at hand as well as the necessary equipment.

The Supervention of Carcinoma.—Finally, a neglected adenoma may become carcinomatous; in fact the majority of carcinomata of the thyreoid that I have seen seem to have been associated with an adenoma, often of long standing, for example, 45 or 50 years. It is generally agreed that at least 1% of adenomata ultimately become carcinomatous, and taking carcinoma of the thyreoid gland as a whole, collected statistics show that 87% to 90% of malignant conditions are associated with adenoma.

The lesson then to be learned is that any adenoma definitely palpable should be removed irrespective of the age of the individual, unless he is very young or very old. But remember in all cases to examine both lobes of the gland, as often there may be one or more nodules not evident from an external examination, and usually set well back in the lower pole, or possibly intrathoracic.

Multiple Cysts and Adenomata.

Multiple colloid cysts (nodular goitre) and multiple adenomata of the thyreoid frequently present a difficult problem, especially if they have grown to any size and the whole gland appears to be involved.

I have frequently removed one, two or more adenomatous or cystic masses which appeared to be discrete tumours in otherwise normal gland tissue, only to find in a few cases recurrence some years later; and it is no uncommon thing to have patients operated on elsewhere to come with second or third recurrences. It is very difficult in these cases to judge the state of the gland proper, and I find now it is wiser to perform a lobectomy for unilateral adenomata, or a subtotal removal if the gland is involved generally, especially when there are multiple colloid cysts. In the extreme cases, in which very little, if any, normal gland is present, I find it best to do a total removal. The most dangerous age period with multiple adenomata or colloid cysts is about the menopause, as so many become toxic, and breakdown of the cardiac mechanism is very frequent. In other cases myxœdema may supervene, whilst now and then one sees those interesting patients showing definite signs of myxœdema with equally positive evidence of thyreotoxicosis. It is this class of patient particularly which makes me believe that the trouble is more likely to be a toxicity of the gland secretion than hyperthyreoidism, since if the trouble was merely one of excessive secretion of thyroxin we should not have coincident myxœdema.

Thyreoiditis.

Primary inflammatory conditions of the thyreoid gland are unusual, and acute thyreoiditis due to an acute infection is rare. One occasionally sees the chronic thyreoiditis known as Reidel's struma, in which there is a generalized stony hardness of the gland without tenderness, the gland being very firmly fixed. It is sometimes difficult to differentiate this condition from primary carcinoma; but the length of history, absence of involvement of lymphatic glands, and the general condition of the patient are helpful points in diagnosis, while, if there is interference with the recurrent laryngeal nerves or any tracheal irritation, carcinoma can be suspected. Most cases of sudden painful tender swellings of the thyreoid are due to hæmorrhage into a colloid cyst.

Malignant Disease of the Thyreoid.

Primary Carcinoma.—Primary carcinoma may be of the proliferative type, rapidly involving the whole gland and very rapid in its course, whereas the scirrhous type remains localized for a long time in one lobe. Lymphatic glands in the adjacent areas of the neck may be involved at an early stage; but the general tendency is for carcinoma of the thyreoid to remain locally malignant for a relatively long time. Most of these tumours are radiosensitive, and even when there is extensive gland metastasis I have known patients to live for many years after deep X ray therapy.

One patient reported last week, who was first operated on in 1917. She had had three or four subsequent operations and much deep X ray therapy. She has had no recurrence for six years now, and one feels that she may have been cured.

Very occasionally one meets with metastasis in long bones; the last two I have seen were both in the lower end of the femur.

Sarcoma.—I have only once seen sarcoma of the thyreoid. It was associated with an adenoma of long standing and was of the large spindle cell variety; the mass was completely encapsuled, and lobectomy was carried out without trouble. The condition was recognized only after pathological examination and must have been of low virulence, as the patient had no recurrence four years later.

Minute Anatomy of the Thyreoid Gland (according to Williamson and Pearce), the Thymus and Parathyreoid.

The Thyreoid.

In the fœtus the epithelium of the thyreoid is arranged in the form of long columns of tissue, straight or coiled. During life the solid core is replaced by colloid or by secretion, which materially alters the microscopic appearance. The simplest conception of the minute anatomy of the thyreoid is that of a collection of "gland units", each of which consists of an endothelium-lined cavity enclosing coiled columns of epithelium surrounded with a basket-work system of capillaries. These capillaries enter the endothelial sac at its hilum and originate from the intralobular vessels. The endothelial sac itself is actually an extension of the perivascular lymph channels which accompany the intralobular capillaries, and it constitutes a lymph space or lymph sinusoid in which lie the epithelial columns of thyreoid tissue.

The lymph sinusoid is by no means a potential space, as there is often to be seen in it a thin, fluid, non-colloid, lymph-like matter, separating the epithelial columns; very often numerous lymphocytes are also found.

Both embryology and comparative anatomy provide evidence in support of such a construction of the thyreoid and its very intimate association with the lymphatic system (*vide* the development of thyreoid *anlage* and the crude thyreoid apparatus of the adult angler fish).

Taking, then, the conception of the "gland unit" as a basis, we find that a collection of such units forms a "lobule", which contains a central lymph channel common to the contained gland units. The only peculiarity of the lobules is that their intralobular blood vessels are simple endothelium-lined vessels, so that arteries and veins are indistinguishable as such.

The lobules of the gland are bounded by an ample fibrous-tissue stroma, in which arteries, veins and lymphatics run to and from the lobules and their gland units. This stroma knits lobule to lobule, thus forming the "lobes" of the gland.

In each lobe there is a clearly defined hilum on the mesial surface, where the inferior thyreoid arteries enter, and these constitute the functional blood supply to the gland units.

The superior thyreoid artery has been found to supply only the interstitial tissue septa and the fibrous capsule of the gland, thus recalling the dis-

tribution of the hepatic artery in the liver. There is, however, a very free "shunt circuit" between the smaller branches of the superior thyreoid artery and the larger surface veins, a fact which has led to much speculation, but for which there seems no adequate explanation. This shunt circuit is responsible for the thrill and bruit found in some goitres.

Far more important than the arterial supply is the lymph vascular system of the thyreoid apparatus. It appears that the relatively few lymphatic vessels which drain to the lymphatic glands in the neighbourhood of the thyreoid gland come only from the interstitial tissue, but the large lymphatic vessels emerging from the hilum have a very different destination and function. These vessels form a "thyreo-thymic leash", which runs down from the hilum of the thyreoid to the upper pole of the thymus in the superior mediastinum, and very often varicosities or pseudo-cysts are to be seen in this leash, depending on the functional activity of the gland. Also in this leash one frequently encounters isolated portions of thymic tissue which may be mistaken for parathyreoids, especially when near the hilum of the thyreoid.

The sinusoidal lymph and migrating lymphocytes from the thyreoid gland can be found in the lymphatic vessels of this thyreo-thymic leash. The study of spread of carcinoma of the thyreoid tends to confirm this free thyreo-thymic connexion, and even as far back as 1832 Sir Astley Cooper drew attention to this connexion between thyreoid and thymus.

The Thymus.

This now brings us to a consideration of the thymus, an organ which most of us seem to forget about, except in relation with so-called *status lymphaticus*.

The thymus is composed of endothelium arranged in nodes or lobules, the individual cells of which also form the walls of a complex aggregate of venous and lymphatic capillaries. The lobules are held together very loosely by interstitial tissue, in which are found the typical Hassal corpuscles. The arterial supply is extremely poor.

The microscopic appearance of the thymus varies widely according to the physiological demands made on it, and shows three distinct phases: (i) lymphocytic infiltration, (ii) granular or lipoidal fat, (iii) vesiculated fat. The last-mentioned phase is the one usually found in the normal adult, and explains the common view that the thymus suffers atrophy in early adult life. The lymphocytic phase occurs when the thyreoid gland is most active, and is found in all thyreotoxic conditions.

The thymus may be looked upon as a tissue which modifies thyreoid secretion and stores any excess.

The Parathyreoids.

I shall not deal with the anatomical variations and peculiarities of the parathyreoids, but I wish to mention their physiological activity as part of the thyreoid apparatus. Like the pituitary body and the adrenals, they are closely associated with the sympathetic nervous system. A second feature is the essentially tubular arrangement of the parenchyma and its variation according to the activity of the thyreoid, as occurs with the thymus. The same type of secretory fluid and lymphocytes may be seen in the lumen of the tubules of the parathyreoid as is seen in the lymph sinusoid in states of secretory activity of the thyreoid.

Thus it would seem that these three gland bodies are closely associated in working towards the execution of some common purpose.

Physiological Considerations.

It has been shown that the normal thyreoid gland shows two distinct phases of activity. The one most commonly seen is that of passive accumulation of colloid; the other is the active process of secretion of a granular non-colloid fluid already referred to. It was difficult to reconcile these phases so long as the simple follicle was regarded as the unit of function; but with the concept of the gland unit of function one could reconcile the changes seen.

Colloid storage is a state of quiescence in all parts of the gland unit, and it seems that before secretory activity can be entered upon the stored colloid must be evacuated, and the epithelial tube returns to the solid fœtal form before it enters on the stage of active secretion.

In the secretory phase a very different picture is presented, as all elements of the gland unit show evidences of activity, even the nuclei and cytoplasm of the epithelium, while the thin granular fluid fills the lumen of the tubules as well as entering the lymph sinusoid. One gland unit can carry out only one function at a time: either colloid storage or secretory activity.

It is worthy of note that colloid is never seen in the lymph sinusoid. In the new-born, secretion seems to be the only product of the gland, and it is significant that at this age the gland is devoid of iodine, and, incidentally, of colloid.

Colloid storage increases up to the age of five years, and from then to fifteen years secretory activity predominates. From fifteen to twenty years of age sex largely determines the picture, the female presenting in the main a secretory phase, while the male tends to colloid storage. After twenty years the female approximates the male type, except during pregnancy and menstruation, when secretory activity occurs. But it is well to remember that storage of this secretory fluid does not occur in the normal gland; it is transferred to the thymus and possibly to other lymph-endothelial tissues. In fact, the pathology of the thyreoid gland appears to be determined by the failure of the tissues to effect a normal reabsorption of the secretion, and it is for this reason I have so stressed the minute anatomy and physiology of the gland units.

Applied Pathology.

We may now briefly consider the morbid physiology of the main types of goitre, although alteration

in the various thresholds of absorption, coordination, utilization or excretion of the thyreoid apparatus gives infinite variations.

Disorders of Secretion.

Primary Graves's Disease.

We shall first consider disorders of secretion, of which primary Graves's disease is the outstanding example, although not the most common in my experience. The whole thyreoid gland in this condition presents the typical adenoid structure in all cases and is distinguished by its freedom from iodine and of colloid, the gland being in a state of intense secretory activity. Secretion produced in the absence of colloid in some parts of the gland cannot exercise a normal physiological effect, probably on account of, as Boothby suggested, a lack of iodine, so its effect on the body tissues becomes pathological.

In these cases the thymus, although enlarged, shows no lymphocytic response, so that the thymic reservoir is not being called upon to function and the secretion is being absorbed as soon as produced. Thus both demand and supply are exalted to a pathological degree, and this rather indicates some factor extrinsic to the thyreoid gland as being the cause of this "adenoid" type of goitre. The early symptoms are definitely those of a true hypersecretion, that is, excess of thyroxin; and if appropriate treatment is instituted in the early stages, recovery in such cases may occur as a result of purely medical measures. Unfortunately, these early cases are not often recognized, and the patient is apt to be classed as neurasthenic. It may be noted here that I regard "hyperthyreoidism" as being an excess of normal secretion.

The second stage in the progress of this condition is the filling of the follicles until they are choked with secretion. The sinusoids become empty and secretion is forced through abnormal channels, that is, direct into the capillary blood vessels. This stage is signalled by an exacerbation of symptoms, and we begin to recognize that a thyreotoxic factor is replacing the hyperthyreoidism. This condition may respond to judicious medical treatment.

The third stage is represented when a diffuse fibrosis of the walls of the sinusoids occurs, rendering normal evacuation of secretion impossible, and it is now taken up direct by the capillary blood vessels. The thyreotoxicosis becomes continuous, and, although responding to medical treatment, is not curable, that is, the gland is not capable of return to the normal.

I feel confident that this third stage can be induced by giving large doses of iodine over long periods, as it produces not only an intralobular fibrosis but a perithyreoid fibrosis, which adds to the difficulties of the surgeon.

At times a slow natural cure will result if the fibrosis persists sufficiently to induce atrophy and exhaustion of the thyreoid gland, always provided the patient can stand up to the thyreotoxicosis in the meantime. One even meets with myxœdema in the very late stages.

Secondary Graves's Disease and Simple Thyreotoxicosis.

Quite distinct from primary Graves's disease are the secondary Graves's disease and simple thyreotoxicosis. Primary Graves's disease is always a disease of early adult life. These secondary types include many varieties and are mostly seen in patients from 25 to 35 years of age and over 45 years; they may follow acute Graves's disease. They are slower in onset of symptoms, which for some time may be those of hypersecretion only, but which gradually give way to the true thyreotoxicosis.

The thyreoid gland in the common type is usually the seat of multiple adenomata or of multiple colloid cysts, although a single adenoma may induce symptoms. In other cases the thyreoid seems small and atrophic, on account of sclerosis from perilobular and periglandular fibrosis. These are the cases in which chronic sepsis has often played a part or the presence of adenomata has over a long period induced fibrotic changes throughout the gland. Colloid, and therefore iodine, is always to be found in parts of the gland; but, owing to fibrosis around the gland unit, it is not capable of absorption. In other areas the typical picture of secretory choking has gradually developed.

Some of these cases go on to some degree of natural cure, as the fibrosis spreads and atrophy occurs, and the amount of actual thyreotoxicosis varies greatly according to the rate of spread and extent of damage through the gland as a whole. Obstruction to the normal absorption of colloid and to the normal outflow of any excess of secretion is the fundamental pathological feature of this condition.

Long-continued financial and domestic worry, with general ill-health, causing undue strain on the normal functioning of the gland, with repeated pregnancies, or even the normal menstrual demand on an overworked thyreoid, seems to account for the greater preponderance of these cases in women.

The explanation of exophthalmos is still a matter for conjecture. In primary Graves's disease it always occurs at an early stage; in the secondary types it is relatively late, and in simple thyreotoxicosis it may never appear at all.

A consideration of this work of Williamson and Pearce, already detailed (*The British Journal of Surgery*, Volume XIII, Number 51, January, 1926, and Volume XVII, Number 67, January, 1930), has provided me with a rational explanation of thyreoid disorders, especially the toxic goitres, and I am in general accord with their thesis, except that in the early stages of secretory disorder a true hyperthyreoidism is unlikely, but if anything a hypothyreoidism or even thyreotoxicosis is more probable. This probably results from incompletely formed thyroxin associated possibly with a lack of available iodine, and instead of the tetra-iodo compound (which is thyroxin) a di-iodo or other

compound lower in the scale is produced, and this may be actually toxic.

Disorders of Colloid Storage.

Colloid goitre shows the inverse picture of the adenoid goitre of acute Graves's disease. There is an entire absence of secretory activity in the gland, and the follicles of the gland unit are distended with colloid, the sinusoids are empty and the lymph channels are not in evidence. This may arise as a relatively acute condition, especially in males at puberty.

In the chronic type the increased storage may go on to such an extent as to cause the formation of colloid cysts. In the earlier stages, especially in the physiological thyreoid enlargements about puberty, there appears to be an actual over-production of colloid, whereas in the chronic type there seems to be a stagnation of the normal turn-over. Both types are suitable for medical treatment, usually by small doses of iodine to supplement the demands of the body tissues, and occasionally some small doses of thyreoid substance to augment the diminished secretory activity.

Operation should be undertaken in the chronic stage only if pressure symptoms supervene or for the cosmetic effect.

One of the most common types of non-toxic goitre and one of the endemic varieties is chronic hypertrophic goitre, associated with some degree of fibrosis through the gland, which interferes with both colloid storage and with secretory activity. A true hypertrophy of the gland ensues, to compensate for the normal demands of the body, and an obvious goitre develops; but the battle seems generally to be a losing one, and these patients frequently become myxœdematous to some degree and seldom if ever become thyreotoxic, as do the patients affected with the chronic vesicular colloid or nodular types. Iodine seldom does them any good and some authorities regard it as actually harmful.

Endemic Goitre.

These last-mentioned types of goitre represent the majority of endemic goitres, which are usually thought to be due solely to iodine deficiency; but McCarrison points out that iodine acts only (or has its major value) in counteracting certain goitrogenic agents, as endemic goitre does not occur in areas where the iodine content of soil and food is low, and these goitrogenic agents do not occur. McCarrison places the factors of causation in four groups:

1. Faulty diet:
   - *A.* Excess of certain substances, for example, fats and lime.
   - *B.* Deficiency of certain substances, for example, iodine, vitamin *C*, vitamin *A*, and protein and phosphates relative to excess of lime.
   - *C.* Goitre-producing substances: cyanogen compounds, as in cabbage.
   - *D.* Insufficiency of antigoitrogenic substances, especially iodine, which largely counteracts all these other dietary factors.
2. Chemical substances, for example: calcium, borax, amines, cyanides *et cetera*.
3. Insanitary conditions, especially long-continued contamination of food and water by human and animal excreta.
4. Infections of a chronic nature, especially tuberculosis.

Classification.

From a consideration of what has been said one can adopt a very simple classification of disorders of the thyreoid gland, and in practice it will be found that all cases can be readily fitted into one or other of the classes.

1. Disorders of secretion.
   - (i) Hyperthyreoidism:
     - (*a*) Physiological.
     - (*b*) Imbalance of the sympathetic.
   - (ii) Primary Graves's disease (adenoid goitre).
   - (iii) Secondary Graves's disease.
   - (iv) Simple thyreotoxicosis:
     - (*a*) Adenomatous (toxic adenoma of Plummer).
     - (*b*) Atrophic (senile type usually).
2. Disorders of colloid storage.
   - (i) Physiological (adolescents).
   - (ii) Colloid goitre (vesicular or nodular).
   - (iii) Chronic hypertrophic goitre (parenchymatous).

Iodine and the Thyreoid Gland.

The administration of 1·0 milligramme of iodine daily in any form has been recommended as being all that is required as prophylaxis against simple goitre, and in respect of this we must remember that the total iodine content of the average normal thyreoid is only 25 milligrammes. The sudden administration of large amounts of iodine often cause the thyreoid to swell and become painful because of the rapid accumulation of colloid and because of an actual fibrositis, intraglandular and periglandular ("iodine thyreoiditis").

Harington argues that we may assume that the iodine of the thyreoid is present entirely in the form of thyroxin and di-iodo-tyrosine, of which the former represents 30% to 40%.

Thyroxin is a tetra-iodo compound, and it seems that in its production in the body tyrosine links up with iodine to form a 3:5 di-iodo-tyrosine, part of which is converted into thyroxin. Thyroxin and di-iodo-tyrosine are linked through other amino-acids and built into the molecule of colloid or thyreoglobulin and so stored in the gland. The true active hormone of the normal thyreoid secretion is probably a peptide containing both bodies. Di-iodo-tyrosine is now on the market and is much lauded in treatment for toxicity; but I find very little difference between it and iodine; also it is very expensive.

As regards thyroxin, the total amount functioning in the body is estimated to be only 8·0 to 15·0 milligrammes, and in the normal person the daily utilization is only 0·2 to 0·4 milligramme. It has been found that 1·0 milligramme of thyroxin increases the basal metabolic rate by 2·8% above the normal, so that in the healthy person the iodine balance is one requiring very delicate adjustment.

On the basis that the normal basal metabolic rate is ± 0, we could estimate that a patient with a

basal metabolic rate of +28 has an excess of 10·0 milligrammes of thyroxin circulating, causing a corresponding increase of katabolism of the tissues. If an excess of thyroxin alone were the cause of symptoms one would expect to see a variation of the clinical phenomena in direct proportion to the basal metabolic rate; but such is not the case, and as a result of my experience I maintain that the basal metabolic rate is not an index of the severity of the condition, but is a useful guide to its progress one way or the other.

One frequently meets with patients in a serious condition with a low or even subnormal basal metabolic rate, just as one meets with others relatively well but with a high basal metabolic rate. The only way I can explain these variations is that there must be some other factor to be considered, and as this factor produces such devastating change in the individual it must be of a toxic nature. Thus it is I advocate the term thyreotoxicosis as against hyperthyreoidism, which connotes an excess of normal secretion.

The minimal daily iodine requirement of the normal thyreoid gland in adults is approximately 45 microgrammes, that is, 0·32 milligramme per week (approximately $^{2}/_{375}$ of a grain).

It is as well to bear these normal needs of the thyreoid in mind when ordering iodine for patients. Lugol's solution consists of 5% of iodine and 7·5% of potassium iodide in water, so that, considering the iodine alone, 0·2 cubic centimetre (three minims) of Lugol's solution contains 9·0 milligrammes (one-seventh of a grain) of iodine, and taking this dose three times a day, the patient receives more in twenty-four hours than the total normal iodine content of the thyreoid gland.

The thyreoid, although taking up iodine avidly, can deal with an excess for a time, but not with a constant overload. The iodine content of the thyreoid is highest in people living on sea borders, and lowest in those living at high altitudes, and it has a seasonal tide, being high in the summer and low in winter. The iodine content of blood under normal conditions is only twelve microgrammes (0·012 milligramme) *per centum*, and the entire iodine content of the blood is less than 1·0 milligramme, but is always increased in the earlier stages of menstruation and in pregnancy.

In thyreotoxic states the blood iodine content is raised, and averages 27 microgrammes *per centum*. In this connexion it may be pointed out that the blood iodine is found in two states: (*a*) organic, which is probably the true thyreoid secretion, and (*b*) inorganic. In toxic conditions the administration of iodine raises the concentration of inorganic iodine in the blood stream, and there is a corresponding decrease of the organic iodine content. It is during this stage that such clinical improvement is noticed in the patient, and it corresponds to a phase of secretory diminution in the gland and an increase in colloid storage. But if the administration of iodine is persisted with over any lengthy period, we find that the organic iodine content of the blood rises and we get what I have called the "hyperiodized patient", that is, there is in the greater part of the thyreoid as full a colloid content as the damaged gland can take; but its secretory phase develops in other gland units, and despite the high inorganic iodine content of the blood the thyreotoxic condition recurs and now will not regress when further inorganic iodine is given. As already mentioned, this overloading of the gland induces an actual thyreoiditis, both intraglandular and periglandular, and this further increases the damage in the gland. It is worthy of note that excess iodine is excreted in the urine and in all thyreotoxic states there is an "iodine diabetes".

Factors in Treatment.

After a perusal of the foregoing it will be seen that in disorders of the thyreoid gland one must be very careful to regulate the dosage of iodine to the needs of the individual. In disorders of secretion, that is, in thyreotoxic states, iodine is usually given in big doses and often for prolonged periods. My experience has taught me that the best results are obtained by small doses for short periods, and usually it should be used only as a preoperative measure, the patient being at rest in bed; for once its beneficial effect is lost one can never get the patient into such good condition again.

In my own practice I seldom give more than three drops of Lugol's solution three times a day; in acute conditions I may give five drops for a few days only, and I seem to get good results. One thing is certain, that my patients do not get iodine thyreoiditis, and so operation is much easier than if there is a brittle gland which will not strip from the surrounding fascia.

I would summarize the uses of iodine as follows: (*a*) as a preventive in districts where endemic goitre is known to occur; (*b*) in selected patients under twenty years of age, suffering from colloid goitre; (*c*) in toxic cases, in the preparation of the patients for operation; (*d*) in cases of true hyperthyreoidism, in small doses for short intervals; and (*e*) in early mild toxic cases in which operation may possibly be deferred, but in minimal doses (two or three minims twice a day, week on and week off) for three months or so.

Almost as important in my mind as the iodine premedication is a preoperative and post-operative alkalinization of the patient.

With the augmentation of the biochemical processes of the bodily tissues in general and the resultant increase in waste products there occurs a diminution in the alkali reserve of the tissues, caused by the great need for buffering the increased acid content of the serum. Thus there exists a tendency to the development of a true acidæmia, which is incompatible with life. I am sure this was a factor in those grave post-operative crises we used to see, and is a factor in the big reactions seen in cases of severe thyreotoxicosis, in which

the patients have not been subjected to a proper preoperative régime.

I have for many years given alkalis both before and after operation. In addition I attempt to increase the glycogen storage of the liver and muscles by giving glucose also before and after operation.

Hyperiodism.

Of the many varieties of toxic goitre that come my way, none is so difficult to treat as the hyperiodized one. Recently I was asked to take over a patient who had been under careful treatment in bed for four and a half months, taking Lugol's solution in doses of 15 to 45 drops a day. "The response in the first few weeks was so marvellous that cure was anticipated, but as the patient was now steadily going down hill surgery was proposed." The record in this direction is that of a woman with a toxic nodular goitre, who had been treated continuously with Lugol's solution in a dose of ten minims three times a day for seven years, while she received intercurrent X ray therapy. She is still in a toxic condition and now desires operation, as auricular fibrillation has supervened and congestive heart failure is commencing.

The only thing to do with these patients is to keep them at rest without iodine for six to eight weeks, and give them alkalis, bromides, vitamins *A* and *C*, and a liberal diet. Then try them on small doses of iodine for a few days only. If no response is obtained, give deep X ray therapy, and in another three months they may be fit for some operative procedure; but it may have to be done in stages.

At times I treat these patients as I do those with severe fibrillation, by removing the thyreoid gland completely. If they show any tendency to myxœdema, it can readily be corrected by a daily dose of 0·09 to 0·18 gramme (one and a half to three grains) of the whole gland thyreoid extract. Again, as with iodine in goitre, when treating myxœdema (or cretinism), always start with minimal doses and work up to the lowest maintenance dose that suits the individual.

X Rays in Treatment.

I consider that only a limited number of thyreotoxic patients are suitable for X ray treatment, namely, those with primary Graves's disease, some with secondary Graves's disease, and the hyperiodized. Where adenomata or colloid cysts exist X rays are relatively useless. This failure to recognize the right types has brought X rays into discredit, and in most cases figures given do not show a true reflex of the value of X ray treatment. Personally I rather favour deep X ray therapy, and find it a valuable adjunct to surgery in the treatment of patients with very severe primary or secondary Graves's disease. Very occasionally one sees an apparent cure; but usually the effect of deep X ray treatment is to reduce the size of the goitre, to inhibit the nervous phenomena and to lower the basal metabolic rate. It has least effect in slowing the heart action and controlling fibrillation, and it is for this reason that operation should be undertaken in two to six months from the completion of the course of deep X ray therapy. During this interval the patient usually puts on a good deal of weight and is much better fitted to stand operation after a short period of preparation. The actual operation is rendered slightly more difficult; but patients as a rule seem to have but a slight reaction, and the results often seem miraculous, except for a persistence of the exophthalmos usually present in these cases, which may diminish somewhat but never disappears altogether.

In some of the very acute cases I also advise deep X ray treatment to the pituitary, as I rather feel that a big part may be played in these cases by the thyreotropic hormone of the anterior lobe of the pituitary gland.

An interesting example of this was a man with acromegaly who developed an acute thyreotoxicosis and goitre. His basal metabolic rate was +64%. He was cured of his intercurrent malady by deep X rays to the pituitary gland only.

Pregnancy.

One is frequently confronted with the problem of a thyreotoxic patient who is pregnant. In my experience patients with acute toxic conditions stand pregnancy badly and they may be classed with those who are at times subjected to operative procedures other than thyreoidectomy, that is, they are liable to develop an acute thyreoid crisis. It is quite safe to perform subtotal thyreoidectomy up to the fourth month of pregnancy in these cases, and it should be advised, as the patients do well and go through their confinement without trouble. In the later stages one can rely only on palliative treatment and hope for the best.

It is remarkable how many cases one sees in which pregnancy has been the determining factor in lighting up an incipient or a quiescent thyreotoxicosis.

Operative Mortality.

As regards toxic goitre in general, the chief factors influencing the operative mortality are: (i) preoperative treatment, (ii) the operative technique, (iii) the post-operative treatment.

(i) *The Preoperative Treatment.*—A high calorie diet is essential, and every effort must be made to control emotional and general nervous instability. Avoid excessive doses of iodine and stabilize the alkali reserve of the body. Do not forget deep X ray therapy as a valuable adjunct in certain cases. Glycosuria, if present, must be controlled, and if cardiac breakdown has occurred necessary treatment in cooperation with a physician must be instituted. Above all, do not be in a hurry to institute operation, and do not proceed with it until the patient is in a satisfactory and stable condition.

(ii) *Operative Technique.*—I regard the time factor in bad risks as very important, and if the

operation cannot be completed in 45 minutes it is wiser to cry a halt and perform a second-stage operation two or three weeks later. It is better to do the operation in two stages than risk a life. An experienced and expert anæsthetist is essential for the bad risks or in the very toxic cases, especially when gas anæsthesia is employed. In low-grade toxic cases a light ether anæsthetic may be employed; but the anæsthetist should never allow the patient to become cyanosed.

Premedication is important. I prefer basal narcosis with paraldehyde or "Avertin" to that induced with barbiturates, which are very uncertain in the bad toxic cases, in which efficient premedication is most needed. I now seldom use local anæsthesia, as with a good gas anæsthesia the patients get on well. Hæmorrhage is a very important factor during operation. It is said that an average loss of ten ounces of blood is immaterial; a loss of up to fifteen ounces is well borne, but when more than twenty ounces have been lost danger sets in (A. R. Short, of Bristol). By very strict attention to hæmostasis I seldom find it necessary to give blood transfusion; but it is wise to utilize this procedure if one is in doubt. Good assistants, prompt decision and manipulative dexterity by the surgeon are important factors. It is wise to use a drain in all toxic cases unless one has had a very wide experience.

(iii) *Efficient Post-Operative Treatment.*—If fibrillation has been a feature of the case an intravenous injection of "Digoxin" should be given before the patient is moved from the theatre. On return to the ward a solution of saline, glucose (10%) and bicarbonate of soda (1%) is given by the rectum until the patient will take fluids freely by mouth. One of the virtues of paraldehyde is that it makes the patient thirsty.

Give oxygen under a tent if any cyanosis appears, and keep the patient quiet with liberal doses of morphine hypodermically. If there is much secretion of mucus one small dose of atropine is permissible.

Hyperthermia is not often met with; but if any signs appear, ice packs must be used; and if it can be arranged a continuous colonic irrigation is valuable. Intravenous injection of sodium iodide has been recommended for post-operative crises; but with efficient preoperative treatment such a state seldom arises.

Operative Details.

As regards the actual operation a few points are worthy of note.

A relatively short transverse incision just overlapping the anterior borders of the sternomastoid muscles and situated rather nearer the upper than the lower pole of the gland is quite sufficient, provided you incise right down to the infrahyoid muscles and then in lifting the flaps divide the deep fascia along the anterior borders of the sternomastoids both up and down. Generally the sternohyoid can be retracted, but it is permissible to divide the anterior fibres of the sternothyreoid if they impede access to the upper pole. Handle the gland as little as possible until the upper pole has been freed. In order to ensure minimal hæmorrhage and to safeguard both inferior and external laryngeal nerves it is essential to incise the pretracheal fascia and work close to the gland itself. After retraction of the infrahyoids work under the lateral aspect of the upper pole with a fairly broad blunt dissector until you can see the carotid sheath well exposed. It helps in this manœuvre if the gland is lifted forward and medially by a blunt dissector held in the left hand. Next, work down medial to the upper pole through the little fascial space there with a blunt dissector and you will be able to pass the instrument under the superior thyreoid leash of vessels and doubly ligate it above gland tissue. Then clamp the upper pole and divide it. This allows you, by grasping the upper pole in the left hand, to roll the gland forward and medially and clamp off the vessels, keeping right on the true gland capsule all the time.

I advise always freeing the upper pole on the opposite side as well, because there is so often a large retrolaryngeal process which should be dislodged, and the whole lobe must be fully exposed before you decide on how much or rather how little should be left. In toxic cases I view with great disfavour the relatively common procedure of a wedge resection from each lobe.

If a pyramidal lobe is present it too should be removed. I concur heartily with Sir Thomas Dunhill's statement that any surgeon who does not remove sufficient of the gland to effect a cure of the thyreotoxic condition is doing goitre surgery a serious disservice.

Total Thyreoidectomy.

It is far wiser to remove too much than too little gland substance, and for the past two years I have in certain cases performed total thyreoidectomy. I reported my first 25 cases at the annual meeting of the Royal Australasian College of Surgeons on March 24, 1938. If too much of the gland is left the patient retains a thyreotoxic state, with its attendant disabilities and risk of cardiac breakdown. If myxœdema supervenes, it is very easily controlled by 0·03 to 0·12 gramme (one-half to two grains) of thyreoid substance twice a day, and I have yet to meet a patient who does not regard this as a very small price to pay for good health.

Owing to the remarkable results achieved in the surgery of thyreotoxicosis with associated congestive heart failure, attention has been directed to the performance of thyreoidectomy in certain cases of primary heart conditions, since by lowering the basal metabolic rate the amount of work performed by the heart is diminished, and it is estimated that the velocity of the blood flow is lessened by 50%. From my experience in total thyreoidectomy I find it takes on the average six to eight weeks for myxœdema to become clinically evident. The lowest basal metabolic rate we found was −28%, and if the dose of thyreoid substance is regulated to keep the basal metabolic rate about −15% the patients look and feel well. In these cases of congestive heart failure total thyreoidectomy gives almost immediate and certainly dramatic relief before the basal metabolic rate can be lowered sufficiently for it to be the main factor. This brings up the question of the relationship between thyreoid and adrenal activity.

It is said that thyreoidectomy does not diminish the adrenal output, and that adrenaline is an important factor in the attacks of pain in *angina pectoris*. Yet thyreoidectomy has given relief from angina, and I find in the severe thyreotoxic cases that all symptoms and signs of hyperadrenalinism disappear after operation. I feel that there is such a very close relationship between the thyreoid and the adrenal that I will not allow adrenaline to be given in any toxic case any more than I would allow atropine or belladonna, as I regard hyperadrenalinism as a factor accentuating thyreotoxicosis.

It seems astounding, in view of the remarkable results obtained in cardiac failure in thyreotoxic states, that the operation of total thyreoidectomy has not been more widely practised or advised. One can only agree with G. H. Pratt in his remark that "only by tottering steps does science advance".

A REVIEW OF THE PROBLEMS OF SUPPURATIVE PETROSITIS AND OF ITS SURGICAL TREATMENT, TOGETHER WITH AN OUTLINE OF THE PROPHYLAXIS AND TREATMENT OF OTITIC MENINGITIS.[1]

By D. G. Carruthers,
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The opinion has been expressed that petrositis is receiving an undue amount of attention. A number of writers in different parts of the world have reported careful macroscopic and microscopic study of the temporal bones of patients who have died of otitic meningitis. The fact that their observations have revealed that quite a high percentage of these patients had suppurative foci in the petrous pyramid, with evidence of direct extension thence to the meninges, must cause us always to consider this possibility in a case in which recovery is not taking place. Lindsay,(1) in a recent review of the *post mortem* findings in 28 cases of meningitis, found otitic infection in 15; in seven of these (47%) the meningeal infection was directly traceable to cells of the petrous pyramid. The degree of prominence given to this complication of middle-ear suppuration cannot be too great, then, if its effect is to cause recognition of this possible extension always to be present in the mind of the otologist. The real problem which arises, and which is the aspect of the condition calling for tempered judgement, is the decision as to when to operate and as to the type of operation to be employed.

The first essential, then, is to arrive at a correct diagnosis. Petrositis is a comparatively late complication of acute *otitis media*, and may become manifest even after a mastoid operation, since it is an extension from the middle ear rather than a secondary development from mastoiditis. Briefly, the symptoms suggesting petrous suppuration are as follows.

The first symptom is pain or aching in the orbit of the same side. The pain tends to be worse in the evening and at night-time, but is more or less continuous. Occasionally aching is also felt in the forehead, in the face and in the parietal region. The peculiar distribution of the pain is stated to be due to tension upon the first and second divisions of the fifth nerve, where these trunks pass across the petrous apex. Some observers state that the peculiar orbital pain is produced by irritation of the greater superficial petrosal nerve. In a few cases pain may be felt in the occiput, and is then thought to be due to threatened involvement of the posterior fossa. Here let me give a word of warning as to pain in petrositis. A period of latency is not uncommon when an escape of pus first occurs into the meninges. The fact that such pain has been present, especially in association with the other symptoms enumerated, should put the surgeon on guard. The fact that the pain has subsided may be but a warning of the explosion to follow.

The second symptom is a profuse or moderately profuse purulent discharge, especially when this persists or returns after the mastoid cells have been exenterated. This always causes the surgeon to suspect some overlooked cell. In a percentage of cases this focus will be found to be in the petrous pyramid; and such a location should always be kept in mind.

The third symptom, paralysis of the sixth nerve (Gradenigo's syndrome), may occur in connexion with apical suppuration; but it is not by any means always evident, and is certainly not so common as used to be taught.

Transient facial weakness and a slight vertigo, with nystagmus, have been reported in many cases which later proved to be of petrositis.

A low-grade pyrexia is commonly present, the temperature ranging from 37·3° to 38·4° C. (99° to 101° F.).

Radiography is of considerable help, especially since Nature has been kind enough to provide two sides for comparison. The pictures, however, must be good ones, taken with a careful technique. In this respect we have yet to enlist the cooperation of the radiographer. In some of the public hospitals the pictures are taken by the technician, who, as yet, may not even have heard of petrosal inflammation. Kopetzky, always bearing in mind the possibility of the development of petrositis, has X ray pictures taken of this region in every patient with *otitis media* admitted to hospital under his care. Should the patient not recover, and should further X ray evidence be required, he has the earlier picture with which to make a comparison.

In a large number of cases clinical evidence of petrositis may be entirely wanting, so that it is only at operation, when the petrosal base is carefully inspected, that fistulæ leading into this part are discovered. Such is a not uncommon explanation of chronic otorrhœa.

[1] Read at a meeting of the Section of Oto-Rhino-Laryngology of the New South Wales Branch of the British Medical Association on July 27, 1938.

The difficult case, of course, will always be the one in which symptoms are indefinite, or that in which meningitis is of sudden and dramatic onset. In such types only time and experience with a number of cases will give to the surgeon that judgement which will enable him to decide whether to wait or whether to embark upon a fairly difficult operation. If he is inclined to wait, careful observations made upon the cerebro-spinal fluid every day or every second day may serve as a guide to the possible advent of intracranial extension and a truly dangerous stage of the disease, which would force the surgeon to undertake immediate interference. In a few cases, too, local meningeal reactions will produce alteration of the limb reflexes, with a contralateral positive Babinski reaction, even before noticeable changes occur in the cerebro-spinal fluid. The condition of the fundi and of the blood furnishes no localizing evidence of petrositis.

In the interpretation of the typical signs and symptoms, sphenoidal sinusitis, superior petrosal or cavernous sinus thrombosis and labyrinthitis may have to be excluded.

While the technique of operation still remains more or less in the evolutionary stage, nevertheless the same criteria of surgical thoroughness must surely be employed as are now followed in the modern process of dealing with mastoid suppuration. I have seen mastoid surgery gradually develop from the simple uncapping of the antrum to the technique (followed by most operators today) of careful probing and searching for every cell which may be diseased, and the ultimate opening out and exenteration of all cells. In the petrous pyramid, however, even more so than in the mastoid process, vital structures, such as the labyrinth and facial nerve, the carotid artery and the dura, lie across the more direct pathways to the deep cells which are likely to be infected. The earliest method of approach to the petrosal cells was to seek and follow up fistulous tracks and diseased cells, especially about Trautmann's triangle.

Let us consider the experience of Kopetzky and Almour[2] in this matter of fistulæ, so that at least we may know where to look for them. In forty-six cases of proven purulent infection of the petrous pyramid the findings were as follows. In ten cases there was a post-labyrinthine fistula; in twenty-one the fistula was anterior to the labyrinth, adjacent to the Eustachian tube; and in four an anterior and a posterior fistula were present. In seven the empyema was closed, there being no fistula. Where fistulæ are found the surgeon may on some occasions still rest content with its exploration and enlargement, unless X ray and clinical observations indicate that there are still other and more deeply seated foci which have not been reached. Even in our experience here in Sydney, however, we have seen instances in which a fistula has led the surgeon quite deeply into the petrous base; yet the hopeful outlook, resulting from the apparently complete drainage of this obvious focus, has met with disappointment. Evidence of failure in completely eliminating the deeply seated infection has become apparent when the lesion has still proceeded from bad to worse, and ultimately to a fatal termination.

When there is no guiding fistula to be found, or when it appears essential actually to reach the apical cells in the petrous pyramid, then a method of exposure has to be sought which is sufficiently direct to give positive drainage and is at the same time a reasonably safe method to employ. An attempt to follow through from behind the labyrinth can reach only as far as the internal auditory meatus, and in any case is not of much value unless fistulæ or cells are actually present in that part of the bone. Earlier attempts to reach the apical cells were made by lifting the dura from the superior surface of the petrous pyramid, and thence attempting to delve into the apex. In a few cases this method was successful, because it so happened that the roof of the apical cells was already necrotic and that an extradural abscess had developed and was able to be drained by this approach. In the majority of instances this method is inefficient in providing subsequent drainage, since the dura soon drops back and blocks any opening which may be made into the apical cells. Many patients with petrositis still have an active labyrinth; and unless suppuration extends into the inner ear cavities there still remains a prospect of good hearing, should recovery take place. The surgeon is loath, therefore, to embark upon a procedure which calls for destruction of the middle ear. Unfortunately, however, the only direct and relatively safe pathway into the petrous apex appears to be that which is available through the anterior part of the petrous base; and to gain access to this the middle ear structures have to be sacrificed.

Eight years ago Kopetzky and Almour[3] suggested a technique which called for drilling through that small area of hard bone anterior to the cochlea and beneath the site of the canal for the *tensor tympani* muscle, adjacent to the tympanic orifice of the Eustachian tube. By this method these surgeons and others, especially in America, did succeed in draining the apical cells in quite a number of cases. The method, however, is hazardous, in that there are no guiding landmarks within the bone to be followed, other than the careful application of previously worked out angles, based upon a study of a large number of temporal bones. In addition, the bone is often very dense and hard. Only one millimetre separates the ascending portion of the internal carotid artery from the basal turn of the cochlea. As the surgeon works upwards, this space may widen out to seven millimetres. That is the maximum space available, even when the work is being done immediately beneath the tegmen. There appears to be very little room for error, then, in this geometrical method. It was apparently in attempting to use this approach that Lempert[4] realized the thinness of the outer bony covering of the ascending portion of the internal

carotid artery lying in its canal in the petrous bone. Having uncapped the artery, he was able to observe that the posterior bony wall of the horizontal portion of this canal was a readily recognizable structure, and one which could be followed and without great difficulty broken down, with the gradual uncapping of the cells of the petrous apex immediately beneath it.

This, then, together with the technique of approach to these parts, is the essential novelty of the Lempert operation. Lempert has devised an anterior incision, through which access is obtained to the bony anterior wall of the internal auditory meatus which forms the partition between that canal and the glenoid fossa. In the Kopetzky and Almour operation this partition is thinned down with a burr. Ramadier first, and then Lempert, suggested its complete removal in order to give a wider access to the anterior part of the middle ear and to the tympanic end of the Eustachian tube. Following inwards, 1·25 centimetres (half an inch) of the root of the zygoma and adjacent squama, and then the anterior and inferior portions of the tympanic annulus and the remainder of the posterior bony wall of the glenoid fossa, are removed, the upper end of the Eustachian tube being exposed. The delicate bony canal of the *tensor tympani* muscle is now readily broken away, the muscle itself is removed, and thence immediately beneath these parts the thin bony covering of the ascending portion of the carotid canal is easily broken through and removed piecemeal. The artery is next gently elevated off the posterior wall of its bony canal, whose outer edge lies presenting in the posterior part of the deep bony opening which has been made by the surgeon. Millimetre by millimetre this bony posterior wall of the horizontal portion of the carotid canal is finally broken down, the apical petrosal cells which lie immediately beneath it being simultaneously exposed.

Lempert, in the full description of his method of dealing with petrous suppuration, describes a routine procedure which aims at the more or less complete isolation of the petrous pyramid and exposure of all cells and of the three cranial aspects of the petrous pyramid. Thus he recommends that in a cellular bone the basal portion of the pyramid, underlying Trautmann's triangle, should be completely exenterated, right down to the superior and posterior semicircular canals. He next removes the *tegmen tympani* and *tegmen mastoideum* and a portion of the adjacent squama. He completely uncovers the lateral sinus. The dura is thus widely exposed and is in turn elevated off the posterior and superior petrosal surfaces, access thus being achieved to any possible necrosis and extradural abscesses in either the posterior or the middle cranial fossa. In the final stages the approach through the anterior incision and carotid region is completed.

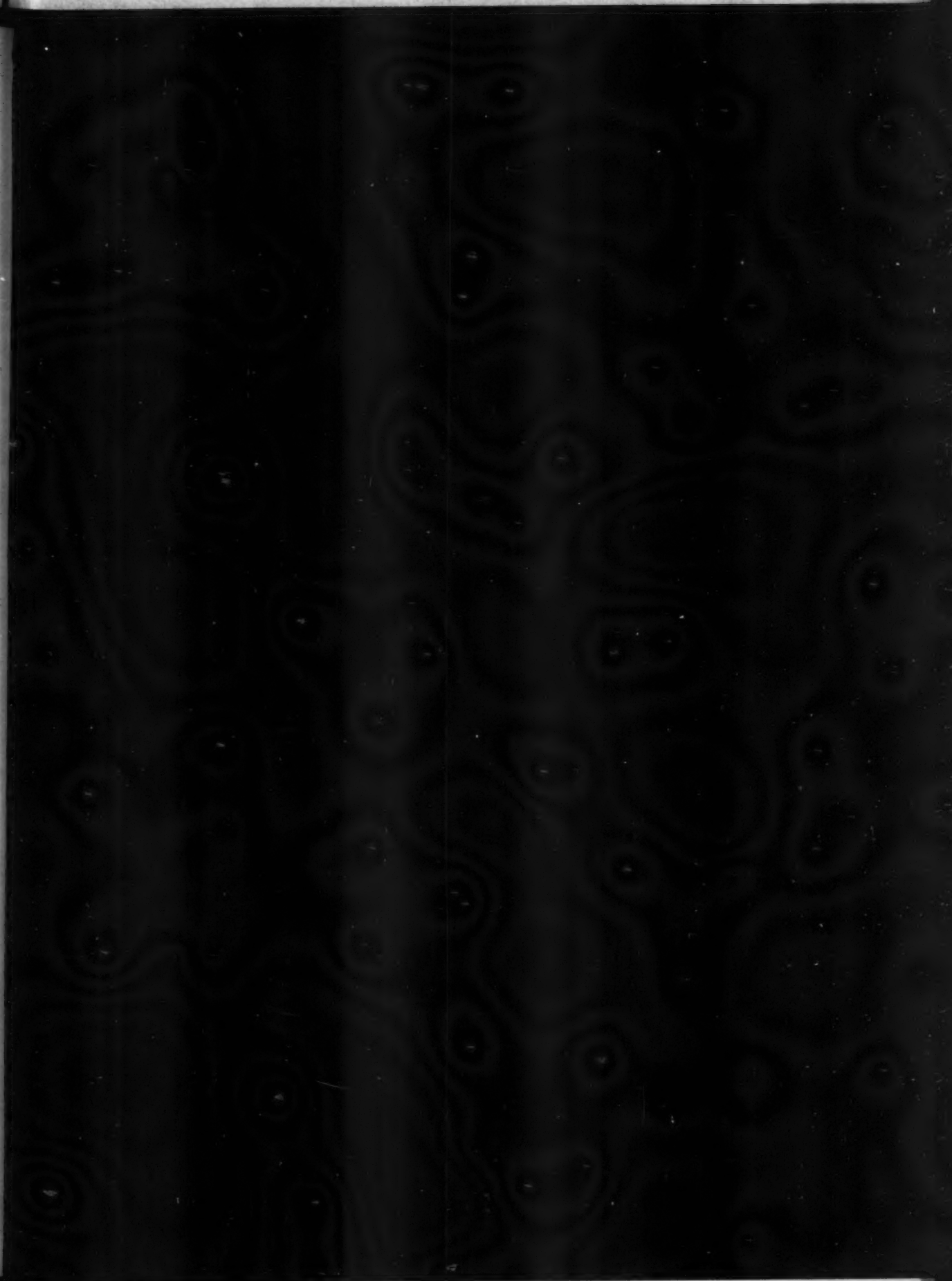
Formidable as the operation seems, and perhaps is, nevertheless it is to be realized that in the majority of cases a simple if not a radical mastoid operation will already have been carried out. It is conceded that many petrosal infections heal spontaneously after the performance of adequate mastoidectomy. Simple mastoidectomy alone will effect this purpose only if the lesion is limited to the posterior labyrinthine area. When the deeper and anterior petrosal area is involved, a radical mastoid operation and the location and drainage of fistulæ still may suffice to effect a cure. It is essential, however, that the otologist should recall the experience of Kopetzky and Almour,[2] as cited above, that in only 25% of their cases was a posterior fistula present, while in approximately 60% the fistula was anterior to the cochlea, and in 15% there was no fistula at all, but a closed apical empyema was present. Evidence of deeply seated petrous suppuration (especially if symptoms persist in spite of simple drainage, and certainly if an increasing cell count in the cerebro-spinal fluid is present) and other changes suggesting threatening intracranial invasion, indicate the presence of a dangerous condition and one which, for want of a simpler approach, must be attacked by this more or less heroic method.

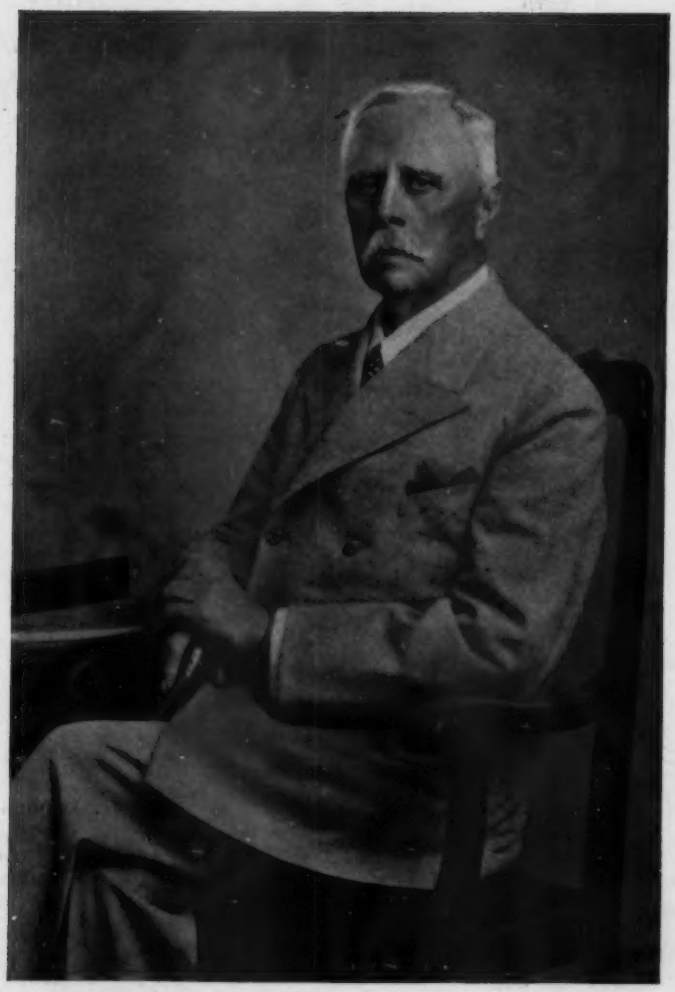
When studying the subject of petrositis and other deeply seated inflammatory processes in the temporal bone, and especially when faced with the possibility of such conditions clinically, the surgeon's thoughts always tend to encompass the problem of meningitis, which may at any time become manifest and convert the patient's condition into a more critical one. Petrosal infection, when included with the widely recognized conditions, such as mastoiditis, attic suppuration, sinus thrombosis and labyrinthitis, probably completes the list of possible penultimate complications of *otitis media* which the surgeon may have to consider in the prophylaxis of otitic meningitis.

The inspiration, especially of the latter part of the paper, is an impression that, rather than being accepted as an unavoidable terminating event in an unsatisfactory case, meningitis should always be in the surgeon's mind, so that he treats the various deeply seated complications of *otitis media* with the feeling at all times that his efforts are really a prophylactic against that condition. With such an attitude the surgeon will always be led to seek for early evidence of this serious development, and he may be encouraged to undertake bolder measures for its prevention.

The problem always is to know when to interfere further, and whether the advance inwards to the meninges has been checked or not.

Since the development of meningitis is always to be feared in the presence of the conditions which have been enumerated, investigators have been seeking carefully for early signs which may indicate the commencement of invasion of the intracranial structures. Considerable progress has been made with the recognition of the value of constant observations upon the cerebro-spinal fluid, based upon the revelations which have come from the study of the cerebro-spinal fluid circulation and its mode of reaction in the presence of infection.





Lund[5] has particularly stressed the value of repeated cerebro-spinal fluid cell counts in deciding when to operate in a case of labyrinthitis. His standards should be equally of value in cases of doubtful petrositis, or when other deeply seated foci are suspected. The normal cell count in the cerebro-spinal fluid is only two to five cells per cubic millimetre. Any elevation, even if of only a few cells, and especially if maintained or increasing, is stated by this authority to be an indication for a labyrinth operation in a case of labyrinthitis, even if the inner ear is not totally inactive. Such changes in the cell count of the cerebro-spinal fluid should be of similar compelling significance in seeking and draining infected foci in the mastoid, the lateral sinus or the petrous pyramid. Kopetzky, however, mentions that a rise of the cell count in the cerebro-spinal fluid up to 30 cells per cubic millimetre may take place even in uncomplicated mastoiditis. In my own experience the cell count has rarely been over five cells per cubic millimetre. For many years now I have been reading of the search, especially by Kopetzky, for still earlier evidence of threatened meningeal invasion than the cell count of the cerebro-spinal fluid. In a recent publication Kopetzky[6] sets out his conclusions, based upon a careful study of the biochemical reactions. He especially stresses a careful comparison between the estimation of lactic acid, carbon dioxide, hydrogen ion and chloride content in the blood and in the cerebro-spinal fluid. He stresses the need for a very speedy examination of the specimens and for careful paraffin capping of the tubes at the bedside, since alteration rapidly takes place in these constituents. It appears that very early in meningeal infection changes take place in the relation between the amounts of these substances present in the blood and in the cerebro-spinal fluid respectively. Let me quote from his paper:

1. There are more chlorides in normal spinal fluid than there are in the blood plasma in the same person. In meningitis this gradient tends to be eliminated, and the spinal fluid and blood plasma are more nearly alike.

2. In meningitis there is a decrease in the bicarbonate content of the spinal fluid, the value often being as low as thirty-three per cent below that of the blood plasma.

3. There is a marked increase in the lactic acid in the cerebro-spinal fluid, and this increase is very much greater than that to be found in the blood of the same patient.

4. The pH of the spinal fluid in meningitis is much lower than either that of the normal fluid or that of the normal blood, and it is also lower than the blood plasma taken from the meningitis case. (Normal cerebro-spinal fluid 7·5. It may drop to 7 or 6·9.)

Suffice for practical application to comprehend that the ratio of lactic acid in the fluid to that of blood is normally about one to one, and in meningitis the lactic acid rises, so that the relation of blood plasma to fluid would be as one to three or more. Summarizing the ionic exchanges, we can say that there is a drop in the chlorides, a drop in the carbon dioxide and a drop in the pH.

The impression gained from reading Kopetzky's paper is that a patient may be very ill, with severe headache and perhaps some signs of a more deeply seated focus in the temporal bone; yet if these laboratory estimations reveal a normal relationship, then we may conclude that the symptoms are arising from the secondary focus in the temporal bone alone. On the other hand, a swing towards the meningeal type of reaction indicates at least that meningitis is imminent. In either case the secondary focus must be speedily and thoroughly evacuated.

Once meningitis has actually occurred the problem of treatment comprises the following four points for consideration:

1. The feeding focus in the temporal bone still must be drained.

2. The offending organisms should be actually determined and typed. Hæmolytic streptococci and the pneumococcus Type III are the commonest offenders. Specific antiserum fortunately has a helpful effect against the streptococcus, but is not so effective against pneumococcus Type III. Fortunately, however, this organism is sensitive to the sulphanilamide drugs.

3. The blood must be constantly fortified. Kopetzky stresses the value of repeated small transfusions of whole blood, given every one or two days. He states that this procedure ranks only second in value to the use of drugs of the sulphanilamide group. He points out that the effect of transfusion in such cases may be due to a restoration of the normal chemical balance referred to above, rather than to any effect upon the hæmoglobin or erythrocyte content.

4. Sulphanilamide preparations should be administered in large daily doses. The usual dose is from 0·2 to 0·25 gramme per kilogram of body weight, given by mouth until the cerebro-spinal fluid has been sterile for forty-eight hours. One-third of the previous daily dosage is given thereafter for seven to fourteen days.

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CATARACTA DERMATOGENES, OR CATARACT WITH NEURODERMATITIS CHRONICA.

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Vogt[1] has described *cataracta dermatogenes* in his last book on slit-lamp microscopy. The book is unfortunately obtainable only in German. Apparently a similar condition was first described by a man named Rothmund as far back as 1868;

later a Russian named Andogsky described its occurrence in a youth and called it *cataracta dermatogenes*.

Ruby K. Daniel,[2] of the Mayo Clinic, describes three cases in an article entitled "Allergy and Cataracts". She suggests that it is an allergic manifestation.

Brunsting,[3] also of the Mayo Clinic, has written an excellent paper on atopic dermatitis of young adults. In ten of his 101 cases cataract was present.

Kugelberg[4] recently made an exhaustive inquiry into the subject of juvenile cataract with scleroderma and neurodermatitis.

In February, 1933, I was consulted, on account of failing vision, by a young man, aged twenty-one years. His face presented most unusual dermatological features, which gave him an unforgettable appearance. This is evidently characteristic of the condition.

At this time I did not realize the relationship that existed between the lens changes which I found and the dermatological condition; but last year, when going through Vogt's clinic in Zurich, I saw a young man who could have been a twin brother of my patient. His facial appearance, on account of the dermatological change, was exactly similar. I immediately made inquiries about his ocular condition, and found that he had complicating cataract. This led to my becoming acquainted with a rare clinical entity, the name of which heads this paper.

The skin disorder of the patient seen by me first in 1933, commenced in infancy and has persisted ever since. He possesses no other disorder that could be regarded as allergic. No other member of his family has had similar manifestations, or asthma, eczema, hay fever or other allergic conditions. His visual acuity in 1933 was $^{6}/_{10}$ in the right eye and $^{5}/_{10}$ in the left eye. Eighteen months later it was $^{6}/_{10}$ in the right eye and $^{5}/_{10}$ in the left eye. In September, 1937, it was $^{6}/_{10}$ in the right eye and $^{1}/_{10}$ in the left eye. This shows that the cataracts are developing very slowly. His visual fields, fundi and refraction are normal.

The skin of his face, lids and forehead was intensely thickened and rough, with some cracks and scabs scattered about as a result of scratching. The lashes and eyebrow hairs were represented by a few scattered, broken-off stubs. The skin was so thick and hard that it could not be picked up. Hard pieces seemed to desquamate, and serous exudate, similar to that seen in eczema, could be seen in places. The cataract was of a definite type. It was entirely anterior and subcapsular, shaped somewhat like a rosette, with definite axial markings. The opacity was extremely iridescent.

The common origin of the epithelium of the skin and that of the lens is said to be the reason for this distinctive clinical type of cataract being associated with the neurodermatitis. The skin condition usually commences in infancy, but the lens change seldom begins until late adolescence. The rate at which the lens changes develop varies very greatly with each individual. Vogt, however, says that in his cases they have developed very rapidly. When he has interfered surgically an intense iridocyclitis has usually followed.

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Reports of Cases.

SIMULTANEOUS MULTIPLE PERFORATIONS OF THE DUODENUM.

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Medical Superintendent, Marrickville District Hospital, Sydney.

Clinical History.

Mr. R., aged fifty-five years, was admitted to the Marrickville District Hospital at 9 p.m. on June 7, 1938.

He gave the history that for the past three days he had had repeated attacks of vomiting after meals, coupled with a dull aching pain in the epigastrium, which somewhat decreased after the vomiting. He went to work on the morning of June 7, and although he felt a few sharp stabs of pain in the epigastrium when bending, he had no trouble until he drank some tea at 11 a.m. He immediately felt a sharp, stabbing pain in the epigastrium which went through to the back. He could not localize it. This pain continued until 1 p.m., when he felt worse, stopped work, and went home to bed. The pain became increasingly severe; at 5 p.m. he had an even more severe attack than the first, which caused him to double up. This was shortly afterwards followed by vomiting and hiccups at frequent intervals. The pain continued until he was seen at 8 p.m. by Dr. R. B. Trindall, who arranged his admission to hospital.

The patient had suffered from indigestion and at times vomiting about two hours after meals for many years. This pain was relieved by more food.

On examination he was seen to be a pale, drawn man in a state of extreme shock. He was obviously in pain and some respiratory distress. He lay immobile in bed, and had frequent hiccups.

Examination of the abdomen revealed no movement on respiration. Muscular guarding and rigidity were present on both sides of the abdomen, most pronounced on either side of the epigastrium. Pronounced tenderness was noted in the epigastrium, somewhat more severe to the right of the middle line. No hyperæsthesia was present and only a very occasional movement was heard on auscultation.

The tongue was dry and coated and the breath was foul. The temperature was 37·1° C. (98·8° F.). The pulse rate was 104 beats per minute and the respirations numbered 40 per minute. No abnormality was detected in the patient's other systems.

A diagnosis of ruptured duodenal ulcer was made, it being thought that at 11 a.m. a leak had occurred, and that this had been followed at 5 p.m. by an acute rupture.

Operation under endotracheal anæsthesia was performed by Dr. R. B. Trindall, assisted by Dr. K. J. Howell. The abdomen was opened by a right upper paramedian incision. Much bile-stained, mucus-like material was found. This immediately suggested a duodenal lesion. It was cleaned away and the duodenum was inspected.

Immediately below Vater's papilla a perforation was found about the size of a threepenny piece. This was surrounded by a large area of induration and inflammation. The rupture was oversown and a piece of omentum was sutured over the opening. Before the abdomen was closed a search was made for other lesions; another rupture was found about half the size of the first and about 2·5 centimetres (one inch) below it. This was

surrounded with much induration, but appeared to be separate from the other ulcerated area. It was easily oversown.

The abdomen was closed, provision being made for drainage; this was terminated after forty-eight hours. Saline solution was given *per rectum* and intravenously for the first twenty-four hours.

The subsequent history was uneventful, the patient progressing after the first few days on a liberal diet of milk and milk foods.

Comment.

The appearance of both ruptures was that of a rupture that had occurred some time previously. This negatived the suggestion that one rupture might have been traumatic at the time of operation.

It seems reasonable to suppose that these two ruptures coincided with the two separate attacks of pain, one occurring roughly at 11 a.m. and the other at 5 p.m. The first probably was only a leak before 11 a.m. Frequent vomiting over the previous three days must have weakened the ulcer walls, which gave way under the strain much about the same time.

I can find in the literature no figures as to the frequency of occurrence of multiple perforations, and most text-books make very little reference to such an occurrence.

Hamilton Bailey[a] makes the following statement:

> Simultaneous perforation of two ulcers has been reported, and we have seen at necropsy a case where one perforated ulcer had been closed satisfactorily and another perforation had been overlooked.

Rodney Maingot[b] states:

> Multiple perforations occurring simultaneously have been described, but are very rare. Multiple perforations are usually found close together. It is more common to find that the base of one large chronic ulcer has perforated at two separate points than that two separate ulcers have perforated simultaneously.

Multiple perforations find no mention in books by Romanis and Mitchener,[c] Rose and Carless,[d] or Rowlands and Turner.[e]

Acknowledgements.

I wish to thank Dr. R. B. Trindall, honorary surgeon to the Marrickville Hospital, for permission to report this case.

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[d] Rose and Carless: "Manual of Surgery for Students and Practitioners", Volume II, 1937.
[e] R. P. Rowlands and P. Turner: "The Operations of Surgery", Volume II, 1937.

Reviews.

OBSTETRICS.

Tweedy's "Practical Obstetrics" is a splendid, well-illustrated book, useful both to the medical student and to the medical practitioner.[1]

The chapter on the administration of anæsthetics during labour is very full; the danger of giving chloroform to patients suffering from toxæmia or *ante partum* hæmorrhage is emphasized, the risk being the development of subsequent changes in the liver.

The description of the conduct of normal labour is excellent, the author stressing the point that control of the fundus during the third stage of labour is unnecessary. He insists that pituitary extract should never be given till the third stage is completed.

Throughout the book there is a great attention to detail. Thus, describing the application of a binder after labour has terminated, the author states: "Before the last pin is inserted the fundus should be contracted and forcibly anteflexed; the binder can then be made slip behind the uterus and keep it fixed and anteflexed."

The chapter on eclampsia is very good. The opinions are expressed that chloroform will not control convulsions and that Cæsarean section is rarely indicated in eclampsia. No mention is made of the ability of "Veratrone" to reduce the blood pressure in this disease. In the section on hydatidiform mole no mention is made of the use of the Aschheim-Zondek reaction in diagnosis.

Placenta accreta is discussed in great detail, the author supporting hysterectomy as the best method of treatment. A table of the differences between the contraction ring and the retraction ring is supplied. The chapter on *ante partum* hæmorrhage is excellent.

[1] "Tweedy's Practical Obstetrics", revised and largely rewritten by B. Solomons, M.D., F.R.C.P.I., F.C.O.G., M.R.I.A., F.A.C.S., and N. McI. Falkiner, M.D., Sc.D., F.R.C.P.I., F.C.O.G.; Seventh Edition; 1937. London: Humphrey Milford; Australia: Angus and Robertson Limited. Demy 8vo, pp. 793, with illustrations. Price: 37s. 6d. net.

DEDUCTIVE ANATOMY.

"A Method of Anatomy", by J. C. Boileau Grant,[1] is hailed by the publishers as something new in anatomical text-books. As far as contemporary text-books are concerned, the claim is justified, but the basic method of linking structure with function maintains the tradition, so long neglected, of Hunter and the Bells. There are six hundred odd pages in the volume. The central nervous system is not described, but there is a useful introductory chapter on dissecting-room procedure, and the book ends with what are really appendices on the autonomic nervous system, the vertebral column, the joints and the bones of the skull. The spelling and style throughout are American, but while frequent repetition of "fiber", "center", "celiac" *et cetera* may desensitize the mind of the reader, such usage as "importantly attached" and "medianly" is apt to offend those with any pretension to a literary conscience. Apart from this, the style is persuasive and lucid. The author has an intimate knowledge and mastery of the subject, coupled with a sincere desire to render the task of the reader as simple as possible. The book is a model of concise writing, which might be copied with advantage by other authors.

The text follows the stages of routine dissection, but the author does not hesitate to leap ahead momentarily to elucidate some point that will simplify the task in hand. Regions are reduced to their bare essential framework upon which the overlying structures are built up in successive, easy stages, until the account of any particular part is complete. Everywhere emphasis is laid upon functional requirements, and the discussion of the mechanics of joints and muscles constitutes one of the most valuable features of the book. Frequent recourse is made to embryological and morphological explanations. In the general description there is sufficient treatment of matters of clinical importance to satisfy the most bigoted "practical" anatomist. The figures are abundant, simple and clear.

Despite its general excellence the book contains statements to which exception must be taken. The gastric nerves derive fibres from both vagi, not from one or other only as is asserted; and Hunt has shown that even the so-called motor portion of the facial nerve includes elements which subserve the function of general sensation, while the ear receives branches from the facial and glossopharyngeal nerves in addition to those from the great auricular, vagus and other nerves. The steadying action

[1] "A Method of Anatomy, Descriptive and Deductive", by J. C. B. Grant, M.C., M.B., Ch.B., F.R.C.S.; 1937. London: Baillière, Tindall and Cox. Crown 4to, pp. 670, with 564 illustrations. Price: 27s. net.

of the flexor muscles in closing the fist may be considered an example of fixation, not of synergism. The account of the *adductor magnus* might have been expanded to include consideration of the oft-occurring *adductor minimus* element; and perhaps some notice should have been taken of Morton's direct measurements of the proportion of body weight that is transmitted through the heads of individual metatarsal bones. The description of the development of the *inferior vena cava* suffers from over-simplification; a slightly more detailed consideration of the supra-cardial veins would have avoided the apparent discrepancy in the statements on different pages that the *vena azygos* passes through the right crus of the diaphragm and that it passes through the aortic opening. In addition it might be doubted whether the gonads undertake such an active migration caudally as is suggested; this movement is probably more relative than real. A surgeon, struggling to cure a femoral hernia, might object to the assertion that Cooper's ligament is a strong fibro-cartilaginous band "through which a needle and thread can get a good grip", while few odontologists would endorse the highly modified "tritubercular theory" of the origin of the teeth, which Dr. Grant advances. In some respects the treatment is uneven. This is especially obvious in the case of the lesser sac, the diaphragm and the female perineum, which are dismissed somewhat summarily; and while the "pyloric vein" of Mayo receives mention, the more important "critical point" of Hartman is denied recognition. The contract to adhere to the Birmingham revision of the Basle *Nomina Anatomica* nomenclature is not strictly kept and there are several lapses into Basle nomenclature and even old terminology. There are several errors in the text, while references to figures frequently prove fruitless. Finally, we observe with regret that the author makes some use of the antiquated method of providing lists of "relations". Nothing is more calculated to destroy interest in anatomy than this practice, which is so contrary to the whole spirit of the remainder of the book that it will come as an unpleasant shock to the otherwise delighted reader.

Notwithstanding these criticisms, the book is the best work of its kind that has appeared in the English language. As a companion volume to dissecting manuals, as a book for rapid revision, or as a stimulus to students who know anatomy reasonably well, it is unsurpassed. Its treatment of the subject will serve to revive an interest in the anatomy of the living subject, which most text-books stifle. It represents a distinct revolt against traditional methods of teaching.

RECENT ADVANCES IN PATHOLOGY.

The third edition of Hadfield and Garrod's contribution to the well-known "Recent Advances" series is a much improved version of a valuable book.[1] A number of books on pathology written in recent years have provided a welcome nexus between the investigations of the laboratory and the clinical observations of the ward. Like Boyd's works, Hadfield and Garrod's book fills this purpose efficiently, besides containing an excellent account of recent work in pure pathology. It would be easy to point to sections that might be expanded with interest; but the authors' difficulty lies in the rejection of matter rather than its inclusion, for to produce yet another text-book of pathology would defeat this manual's primary object. The authors have cast overboard some sections included in the earlier editions, and while lightening the old cargo have added more valuable freight in the form of new sections and expansions of the old. The new first chapter on the subject of resistance to infection, with its condensed account of the conflicting views on allergy, especially in relation to common clinical problems like tuberculosis and nephritis, is particularly useful.

The application of the functions of the reticulo-endothelial system to clinical states is described in another new chapter of exceptional interest. The advances in cancer research and in the study of deficiency diseases have been brought up to date. The chapters on endocrine disturbances have been in parts compressed, but are still quite adequate. Perhaps the authors would have been wiser to omit these altogether, since there is already an excellent "Recent Advances" volume devoted to endocrinology alone. The experimental work on nephritis is well and fairly presented, although it is possible that the pendulum of interest may yet swing back to the changes in the extrarenal tissues in nephritic disease, and that the histological classifications and criteria of the pathologist in nephritis may assume less importance as time goes on. What is required in a book like this is a readable and compact presentation of the relevant recent work in the various fields, treated without undue bias and written with discrimination and some critical force. This third edition fulfills this requirement. Like its predecessors it is well printed and well illustrated.

[1] "Recent Advances in Pathology", by G. Hadfield, M.D., F.R.C.P., and L. P. Garrod, M.A., B.Ch., F.R.C.P.; Third Edition; 1938. London: J. and A. Churchill Limited. Large crown 8vo, pp. 432, with 65 illustrations. Price: 15s. net.

PHYSIOLOGICAL CHEMISTRY.

The eleventh edition of "Practical Physiological Chemistry",[1] by P. B. Hawk and O. Bergeim, writing in collaboration with B. L. Oser and A. G. Cole, follows six years after the tenth edition. In this volume, which is covered with a special washable binding, several chapters have either been rewritten or revised by co-opted authors, and we again notice a serious attempt by the authors to keep abreast with all recent advances in biochemistry.

There has been no substantial growth in the size of the book, and the general character and standard remain of the same high order as in previous editions. Some of the subject matter, for example, the chapter on the endocrine glands, is so condensed as to be practically epitomized, but this has apparently been done to keep the size of the book within bounds.

Much that was written in our review of the tenth edition may be said with equal truth of the present volume. The work is still amongst the best of its kind in the English language. It will take its place proudly on the library shelves of all laboratory workers in biochemistry and will be the guide of students of medicine and the allied sciences in universities where the standard of biochemistry is high.

[1] "Practical Physiological Chemistry", by P. B. Hawk, M.S., Ph.D., and O. Bergeim, M.S., Ph.D., in collaboration with B. L. Oser, Ph.D., and A. G. Cole, Ph.D.; Eleventh Edition; 1937. Philadelphia: P. Blakiston's Son and Company Incorporated. Medium 8vo, pp. 990, with illustrations. Price: $8.00 net.

BACTERIOLOGY FOR DENTAL STUDENTS.

Mr. Arthur Bulleid, the author of "A Text-Book of Bacteriology for Dental Students",[2] has done two things and done them well. He has provided progressive instruction in laboratory technique and presented an excellent review of general and dental bacteriology. All medical and dental students should find this compact volume a lucid aid to their studies, whilst for graduates it might well serve as a quick reference volume. The bacteriology of the oral flora is described in fine detail. A section on special mouth bacteria might well serve as an introduction to an intensified study of this important subject. Mr. Bulleid's recognized standing gives the book high authoritative value, and its wide appreciation is assured.

[2] "A Text-Book of Bacteriology for Dental Students", by A. Bulleid, L.R.C.P., M.R.C.S., L.D.S.; Second Edition; 1938. London: William Heinemann (Medical Books) Limited. Demy 8vo, pp. 224, with illustrations. Price: 15s. net.

The Medical Journal of Australia

SATURDAY, OCTOBER 15, 1938.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

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THE VALUE OF OXYGEN IN THE TREATMENT OF PNEUMONIA.

It would seem a parade of the obvious to state that the use of oxygen in the treatment of pneumonia has caused a decrease in the mortality of that disease, but actually only a few critical surveys of the subject have been made. It must be admitted that oxygen is often used only half-heartedly. It is no exaggeration to say that occasionally a delicately fine catheter conveys a trickle of oxygen into the nasal cavity of a patient and only for a few minutes in each hour; this therapeutic gesture, though well-intentioned, is futile. We should remember that the oxygen saturation of the arterial blood in pneumonia is frequently decreased and that it is the peripheral circulation rather than the actual cardiac mechanism that first shows clearly this state of affairs. If scepticism exists among clinicians as to the value of oxygen therapy in this disease, it is declared by G. H. Faget and W. B. Martin to be due to the misuse or abuse of this therapeutic aid.[1] These writers point out that oxygen is used chiefly for seriously ill and moribund patients, a group that naturally shows a very high mortality rate. For the past five years these investigators have employed oxygen therapy as a routine measure in the United States Marine Hospital, Norfolk. As soon as a diagnosis of pneumonia was made they began the administration of oxygen and maintained a concentration of 40% to 60% in a tent throughout the active period of the disease. The patient was informed at the outset that he would live in the tent during his illness and was assured that this measure would be a factor in his rapid recovery. All care was taken that leakages would not occur and a flow of six litres per minute was employed; this amount was found to ensure the necessary concentration of the gas. The authors made frequent gasometric tests to determine whether this optimum percentage was being maintained; they found that the usual cause of a lowered percentage was a leaky tent. The tent used was one with a blower, which circulated the gas through a cooling and drying device chilled by cracked ice; at first soda lime was employed to absorb the excess of carbon dioxide, but it was later abandoned on account of cost. The percentage of carbon dioxide in the tent was found not to exceed four when the oxygen flow was six litres per minute, and if the oxygen flow was increased to eight or ten litres per minute the carbon dioxide did not rise above 1·5%. This modification of the tent method is probably an advantage, although Faget and Martin apparently do not follow Henderson in his advocacy of "Carbogen" in the treatment of pneumonia. As a matter of fact a tent that contains pure oxygen but allows a concentration of carbon dioxide within safe limits is rather an advantage, for the price of oxygen is considerably less than that of oxygen to which carbon dioxide in a fixed proportion, such as 5%, has been added.

Faget and Martin cannot substantiate all the claims made for the oxygen therapy of pneumonia. For example, they cannot demonstrate a constant lowering of temperature, pulse rate and respiration rate after this treatment, nor do they find that the course of the disease has been shortened. But they note that the patient becomes quieter and less inclined to delirium, that his breathing is easier, that cyanosis is lessened, and that he sleeps more restfully. Over the period of five years of their

[1] *Annals of Internal Medicine*, July, 1938.

study they collected figures of a series of cases of proved pneumonia in men who were either seafarers or employed as coastguards or in a similar naval capacity. The majority of the patients were between twenty and fifty years of age. The response to oxygen therapy was compared with the results of the treatment of pneumonia in similar groups during an equivalent period previous to the present investigation. All other factors, including nursing and general care, were identical, and serum therapy was not employed. Of 49 patients suffering from lobar pneumonia who were treated without oxygen during a period of four years, 16 died, a mortality rate of 32%; during the next five years 64 were treated, of whom 12 died, a mortality rate of less than 19%. The corresponding figures for bronchopneumonia revealed that of 38 patients treated over the control period, 11 died, while seven deaths occurred among the 42 patients over the later period, the mortality percentages being 29 and 17 respectively. Interesting figures of the treatment of pneumonia in other hospitals in Norfolk are quoted by Faget and Martin for comparison; these show an amazingly high death rate where oxygen was used, especially where the duration of its administration was brief. The reason for this is that oxygen was largely reserved for desperate cases of pneumonia and, as Faget and Martin remark, "was often a farewell gesture rather than a well-conceived therapeutic effort".

Faget and Martin's communication indicates that a definite lowering of the mortality rate of lobar and bronchial forms of pneumonia occurs when oxygen is used continuously throughout the illness. Perhaps in the ordinary run of practice a physician of experience may assume the responsibility of declaring that a given attack of pneumonia is mild enough to make the administration of oxygen unnecessary. But pneumonia is a treacherous disease, and even if oxygen is not cheap, life is precious. Certainly it should be more generally recognized that if oxygen is needed at all, it should be given early and continuously in order that its full therapeutic value may be attained. We commend to our readers' attention an article by Professor Whitridge Davies appearing in this number of the journal, on the physiological considerations of oxygen lack. No form of treatment is scientifically sound unless it is based on a proper conception of the fundamental principles involved.

Current Comment.

CEREBRAL LESIONS AFTER NITROUS OXIDE ANÆSTHESIA.

It is generally considered that nitrous oxide is one of the safest of all general anæsthetics; but safety being, of course, at best a relative term, untoward results occasionally have been reported. It must be remembered that the patients to whom nitrous oxide gas is administered are not infrequently ill-suited to any type of anæsthesia. This fallacy in statistics demonstrating the safety or danger of various anæsthetics is, of course, well known. Nevertheless, it is a fact that patients have suffered from respiratory or cardio-respiratory failure while under anæsthesia with nitrous oxide and oxygen, and while some have recovered, others have died. Of those who survived, some have presented a disturbance of the nervous system of the extrapyramidal type, some have manifested psychotic changes, and others have recovered completely.

These accidents are discussed in an article by Cyril B. Courville, who has had an opportunity during the last five years of studying untoward manifestations in a number of patients after nitrous oxide anæsthesia.[1] He states that of the patients who died, all remained in coma, some had frequent convulsions, and most of them succumbed in a state of hyperthermia. The survival period varied from 36 hours to 26 days. It is naturally of great importance that careful histological examination of the brain should be carried out when death ensues, and this has been done by a number of workers, who agree in finding characteristic areas of necrosis in the cerebral cortex and in the region of the lenticular nucleus. The cause of these changes has been the subject of disagreement, some authors maintaining that they were due to the toxic action of nitrous oxide. Courville has reviewed his material to try to arrive at the truth of the matter.

First, he discusses the mode of action of nitrous oxide and raises the question of how much of its effect is due to asphyxia. Here he remarks that from the physiological standpoint nitrous oxide does not behave entirely like a pure asphyxial gas, inasmuch as the pulse of the anæsthetized patient is not so slow, the convulsive movements are less pronounced and there is a rather more specific action on the centres of respiration. It should be noted also that the specific narcotic action of the gas is observed even when it is diluted with oxygen and disappears when an inert gas like nitrogen is sub-

[1] *Annals of Surgery*, March, 1938.

stituted. It would therefore appear that nitrous oxide has a distinct narcotic effect *per se*. There seems good reason to believe, however, that the degree of anoxæmia associated with nitrous oxide anæsthesia is always considerable, unless special means are taken to combat it, a fact well known to anæsthetists. On experimental grounds Courville states that it seems clear enough that the actual narcotic effect of nitrous oxide is not really important so far as any serious or fatal effects are concerned. He reviews briefly the three types of anoxæmia recognized by physiologists, namely, the anæmic, which is self-explanatory; the stagnant, due to a slowing of the circulation as occurs in cardiac failure; and the anoxic type. This last, being due to the lowering of the tension of oxygen in the blood, is the most serious of all, and with it there is usually a coincident lowering of the tension of carbon dioxide. This in its turn decreases further the rate of dissociation of hæmoglobin, so that oxygen which is present is even less readily available to the tissues. Courville points out that the occurrence of this anoxic type of anoxæmia in nitrous oxide anæsthesia may be accentuated by mechanical defects in the apparatus and obstruction of the respiratory passages. He comments further on the mild and transient symptoms which may occur during this form of anæsthesia and which are not, of themselves very important, provided they do not progress to the point where they may provoke more grave symptoms. Thus, even slight degrees of cyanosis, respiratory irregularities and muscular twitchings during anæsthesia should be seriously regarded. In the recovery period stupor or restlessness of the patient may also be observed. When serious results follow, such as psychotic symptoms and signs of extra-pyramidal damage, the manifestations are due to necrosis, and, in Courville's opinion, the necrosis is probably the result of asphyxia not due to the toxic narcotic action of the gas. Experimental work indicates that the earliest effect is seen in the interstitial tissue about the pericellular spaces as well as in the nerve cells, a change more characteristic of asphyxia than of narcosis. The necrotic areas strongly resemble those produced experimentally by temporary ligature of the larger arteries of the brain; such lesions are not found after narcotic poisoning.

It will, of course, occur to those interested in carbon monoxide poisoning that lenticular degeneration is a well-known sequel of this condition, and in this case we have no reason to believe that the cause of the lesion is any other than simple asphyxia. Courville draws this parallel in the case of nitrous oxide anæsthesia. His article contains a number of photomicrographs showing the nature of the damage in the central nervous system, and he also discusses the possible factors concerned in the clinical manifestations. His conclusion would seem to be justified that this type of cerebral damage is due to asphyxia or to a prolonged accumulative anoxæmia. If the lesions are caused by asphyxia, cardio-respiratory failure has been a most important factor, and if by anoxæmia, other predisposing factors might be some toxic state of the tissues such as occurs in alcoholism, and mechanical or other faults in the technique of the administration. These considerations in no way reflect on the general safety of this excellent anæsthetic agent, but they serve to emphasize the point that safe administration requires an efficient technical equipment and high degree of personal skill; and therefore in a difficult or dangerous case the anæsthetist needs to have before him a clear picture of the physiological processes at work.

TREATMENT OF MARASMUS WITH SUPRARENAL CORTICAL EXTRACT.

According to Marriott the outstanding metabolic defects in marasmus are an inability of the body to utilize food and a negative nitrogen and mineral salt balance. Marriott's statement and the fact that the phenomena mentioned are constantly found in Addison's disease led W. Alexander Hislop to hope that benefit might accrue from the administration of active extracts of the adrenal cortex to marasmic infants. The results of his investigations are now published.[1] Marasmus is a final state of the body produced by multiple causes. Ill feeding, latent infections and constitutional defects are all common precursors. Discussing the part played by infections, Hislop points out that the adrenal glands play an important role in antibacterial and antitoxic defence. Hyperæmia of these glands and hæmorrhage into them are not uncommonly found in infants who have died from intestinal intoxication.

At the commencement of his investigation Hislop gave a series of marasmic babies what subsequently appeared to be too small a dosage of suprarenal extract with equivocal results. Fourteen infants, after a suitable "control period", were then treated with daily injections of "Eucortone" in doses of one minim to each two pounds of body weight. The diet and general supervision of the children were unaltered throughout, and all were supplied with a constant amount of a preparation containing vitamins *A* and *D*. Of the fourteen children, eleven gained more weight during the period of "Eucortone" administration than during the control period. When exhibition of the drug was interrupted the ascending gradient of the weight curve was deflected to the horizontal level, but resumed its upward trend on recommencement of the therapy. Of the three infants who failed to respond, one was suffering from *otitis media*, one died after making no progress, and the third left hospital too soon to allow of an adequate assessment of results.

The investigator is careful to point out that he does not consider that all forms of marasmus are due to adrenal deficiency. He merely asserts that an ultimate defect of adrenal function develops, and that correction of this often leads to a gain in the infant's weight. Thereafter the natural processes of recovery consolidate the cure.

[1] *The Lancet*, August 6, 1938.

Abstracts from Current Medical Literature.

GYNÆCOLOGY.

Disgerminoma of the Ovary.

Emil Novak and Laman A. Gray (*American Journal of Obstetrics and Gynecology*, June, 1938) present a study of seventeen cases of disgerminoma of the ovary. They believe that the four "special ovarian tumours", namely, the granulosa-cell carcinoma, the arrhenoblastoma, the disgerminoma and the so-called Brenner tumour, offer a far more favourable prognosis than does ovarian cancer in general. The disgerminoma, also known as large-cell carcinoma or embryonal carcinoma, arises from cells dating back to the undifferentiated phase of gonadal development; and, as would be expected, an exactly similar tumour, the seminoma, occurs in the testis. Unlike the feminizing granulosa-cell carcinoma and the masculinizing arrhenoblastoma, the disgerminoma exhibits no endocrine activity, being made up of sexually indifferent cells. It is often observed in sexually underdeveloped or pseudohermaphroditic individuals; but the authors state that it has nothing to do with the production of these sex abnormalities, which persist even after removal of the tumour. The disgerminoma is preeminently a tumour of early life, thus justifying the name of "*carcinoma puellarum*". The authors believe that while it is undoubtedly a malignant type of tumour, there are pronounced variations in the degree of malignancy of individual tumours. In their opinion the outlook is very favourable when the tumour is unilateral, with intact capsule. In their series, nine of ten patients with such tumours have remained well after operation. The results were much less favourable when the capsule had been broken through, with extensive infiltration of surrounding organs, and perhaps metastases. The microscopic recognition of disgerminomata, according to these authors, is usually very easy. Both the cell type and the general architecture of the tumour are very distinctive. The large, round, ovoid or polygonal cells responsible for the name "large-cell carcinoma" are characteristically arranged in alveoli or nests. There is much less variation in the histology of these tumours than in the histology of granulosa-cell carcinoma or of arrhenoblastoma.

The Ætiology of Thrombosis and Embolism.

In a paper read before the International Congress of Obstetrics and Gynæcology, at Amsterdam, D. Dougal (*The Journal of Obstetrics and Gynæcology of the British Empire*, June, 1938) discusses the ætiology of thrombosis and embolism. After reviewing all the literature, and from his own observations, Dougal is of the opinion that tissue trauma and sepsis are the two primary factors concerned, and as they frequently coexist in cases of thrombosis, it is difficult to decide which is the more important. Slowing of the circulation is so important a predisposing cause of thrombosis that it is almost a *sine qua non*. Changes in the blood, which cause an increased susceptibility to thrombosis, occur during the puerperium and after surgical operations; but these are insufficient actually to cause thrombosis without the presence of one of the more important factors.

Post-Operative Exercises as a Preventive of Thrombosis.

W. F. Shaw and C. E. B. Rickards (*The Journal of Obstetrics and Gynæcology of the British Empire*, June, 1938) give the result of investigations of two series of patients operated on by Shaw. The first series consisted of 3,618 patients at Saint Mary's Hospital, and the second series of 1,635 patients at the Royal Infirmary, Manchester. At the Royal Infirmary the sister in charge of the ward had instructed the patients in regular exercises three times a day. These exercises consisted in lifting the arms above the head twenty times and lifting each leg the same number of times whilst lying on the back. Embolism occurred only once in the series of patients who were instructed in exercises, and eleven times in the control series. It was thus shown to be five times more common in patients in the control series. All these patients were operated on by the same surgeon and the after-care was practically identical, with the exception of routine exercises.

The Experimental Production of Ovulation in the Human Subject.

E. Davis and A. K. Koff (*American Journal of Obstetrics and Gynecology*, August, 1938) describe their work on the experimental production of ovulation in the human subject. Up to the present it has been impossible to show any follicular stimulation in human beings by administration of anterior pituitary-like substances extracted from the urine. Cole and Hart, in 1930, observed that for a limited period the blood of pregnant mares contained a gonadotropic substance in high concentration. The maximum concentration occurs at about the seventieth day of gestation and this substance never appears in the urine. It is not ultrafiltrable through collodion membranes. When the mare's serum containing this substance is injected into immature animals it produces follicle growth. Selecting patients who were to be subjected to laparotomy, the authors gave intravenous and intramuscular injections of this gonadotropic hormone obtained from the serum of mares, and they were able to show by photographs taken during laparotomy or by microscopic examination of ovarian tissue, or both, that definite ovulation took place as a result of these injections. This hormone has been isolated in an advanced state of purity and preparations of it were injected intravenously and intramuscularly without harm. In the biological effects it produces, this gonadotropic hormone resembles extracts and implants of the anterior lobe of the hypophysis, but differs chemically from all other gonadotropic substances heretofore studied.

Spinal Anæsthesia.

R. Hellman (*Deutsche medizinische Wochenschrift*, July 8, 1938) describes his experience in the use of a new preparation of "Pantocain" for spinal anæsthesia in gynæcological and obstetrical cases. He emphasizes the need for the use of sedatives, such as "Veronal" and "Luminal", for several days beforehand when possible. One and a half hours before operation 0·00006 gramme of scopolamine and 0·02 gramme of morphine are given, and the dose of scopolamine is repeated thirty minutes before operation. Morphine is omitted in cases of Cæsarean section because of the effect on the fœtal heart. Unless the patient is very excitable, no further anæsthetic is required to permit insertion of the spinal needle. The author believes that an injection of "Cardiazol", by preventing collapse, assists in extending the effect of the spinal anæsthesia. Ten milligrammes of "Pantocain" are dissolved in four or five cubic centimetres of the spinal fluid. The resultant 0·2% solution should be slowly injected into the spinal canal, between the second and third lumbar vertebræ. Complete anæsthesia follows within fifteen to twenty minutes.

The Value of Blood Examination in Gynæcological Conditions.

K. Hollósi and J. Martzy (*Monatsschrift für Geburtshülfe und Gynäkologie*, May, 1938) discuss the value of the blood count and of estimation of the sedimentation rate in the differentiation of various gynæcological diseases. In the acute stage of ectopic pregnancy there are pronounced anæmia, leucocytosis, and a sedimentation rate reading of 22 millimetres within one hour. In the subacute stage, corresponding to tubal abortion, a higher erythrocyte count, lower leucocytosis, with an increase in lymphocytes and a sedimentation rate of 44 millimetres per hour are present. The chronic stage is marked by a fairly normal erythrocyte count, pronounced leucocytosis and an increase in the sedimentation rate to 65 millimetres per hour. In acute and chronic inflammatory lesions there are definite changes in the blood count and sedimentation rate; but in displacements of the uterus these

tests afford no information. In *metropathia hæmorrhagica* anæmia is present. Fibroid growths are accompanied by anæmia only when hæmorrhage is a prominent symptom. In cases of vesicular mole, if the sedimentation rate remains high after expulsion of the mole, the formation of chorionepithelioma should be considered.

OBSTETRICS.

The Determination of Œstrin in the Urine of Normal and Toxæmic Patients.

J. E. Savage and L. H. Douglass (*American Journal of Obstetrics and Gynecology*, July, 1938) publish results obtained by the use of a quantitative chemical test for œstrin in the urine in the last three months of pregnancy in normal and toxæmic patients. A modification of the Schmulovitz and Wylie tests was used for the detection of œstrin in the urine. It was found that there was a lowered level of œstrin excretion in the urine in a series of twenty-six patients with preeclampsia and also in twenty-six patients with chronic nephritis complicating pregnancy. Fifty-eight patients in the last trimester of pregnancy were carefully studied. Œstrin excretion expressed as a "ferric chloride number" gave an average of 87·16 in six normal cases, of 47·05 for twenty-six patients with chronic nephritis complicating pregnancy, and of 40·03 for twenty-six patients with preeclampsia. Nine patients were given 10,000 international units of "Theelin" intramuscularly on three successive days. The authors were unable to find any improvement in those patients to whom "Theelin" was administered.

The Ætiology of the Toxæmias of Pregnancy.

Working in the Harvard Medical School, M. B. Strauss contributes an observation on the primary role of the plasma protein in conditioning water retention and œdema formation in normal and toxæmic pregnancy (*The American Journal of the Medical Sciences*, July, 1938). In previous papers the importance of water retention in toxæmia of pregnancy had been emphasized. It was shown that toxæmic manifestations might be relieved by a decrease in water retention or increased by the induction of further water retention. The author states that the chief factor responsible for water retention in pregnancy is a lowering of the colloid osmotic pressure of the plasma due to hypoproteinæmia. He believes that there is no necessity to consider a hormonal or renal disturbance in the water retention of pregnant patients. In an investigation of twenty patients it appeared that the plasma protein level was the primary and most important factor. On admission to hospital the patients were placed on house diets without restriction of any sort; salt and water were permitted, no sodium bicarbonate was given, and no saline cathartics were administered. After the preliminary period of observation was completed each patient received 1,500 cubic centimetres of skimmed milk for five days. The percentage loss of weight for each patient was correlated with the calculated colloid osmotic pressure of the plasma proteins. There appeared to be a close relationship between these two sets of observations. All the patients lost weight. One patient, in whom a low osmotic pressure of the plasma proteins was observed, lost only 3% of her body weight on the skimmed milk régime, and continued to have œdema, hypertension and other toxæmic symptoms. Labour was induced. Six weeks later her weight was 17·2 kilograms less, œdema was absent, her blood pressure was normal, and her urine free from albumin. It would appear that in the absence of anæmia, congestive heart failure and acute glomerulonephritis, water retention in both normal and toxæmic pregnancy depends essentially on the level of plasma proteins and may be influenced, as in animals, by changes in the intake of electrolytes.

Rupture of the Symphysis during Labour.

K. Kimmig (*Monatsschrift für Geburtshülfe und Gynäkologie*, April, 1938) presents an exhaustive survey of the literature concerning rupture of the *symphysis pubis* during labour. He draws attention to the frequency of physiological softening of the joint during pregnancy, which is associated with pain, difficulty in walking and a general sense of instability. The normal space of four or six millimetres at the joint may be increased to ten or twelve millimetres in the latter months of pregnancy. Actual rupture of the symphysis is generally associated with difficult delivery by high forceps applied in Walcher's position, assisted by suprapubic pressure. The author emphasizes the important fact that the injury may be overlooked, because pain is often complained of in the hip and not at the pubis. The legs are found to be rotated outwards and abducted, and any movements cause pain. Œdema of the labia and the formation of a hæmatoma are later clinical signs. Treatment consists in the application of some form of tight binder. Recovery generally ensues within a few weeks. During subsequent pregnancies a belt should be worn.

The Relation of Pituitary Oxytocin to Parturition.

C. Fisher, H. W. Magoun and S. W. Ranson (*American Journal of Obstetrics and Gynecology*, July, 1938) do not endorse the view that parturition can take place normally in the absence of the hypophysis. During the course of an investigation of the relation of the so-called hypothalamico-hypophyseal system to *diabetes insipidus*, certain observations have been made which throw doubt on the conclusion that the hypophysis, and more especially its neural lobe, is not involved in parturition. The authors have found that experimental *diabetes insipidus* can be produced in the cat and the monkey by placing lesions in the anterior hypothalamus in such a position that they interrupt bilaterally the supraoptic component of the hypothalamico-hypophyseal tract. *Diabetes insipidus* was produced in eighty-five cats, seven of which were in various stages of pregnancy at the time of operation. All of these animals went to term, but all developed disturbance of the delivery mechanism. Four of them died in the course of a greatly prolonged labour; three of the seven animals managed to survive parturition, but only after a protracted period of labour; two died a few days after delivery. The authors believe that this experiment supports the theory that pituitary oxytocin plays a role in the commencement of labour. It would seem also to lend weight to the theory that labour commences under the influence of increasing amounts of œstrin, the uterus becoming more and more sensitive to oxytocin.

Hormonal Diagnosis of Pregnancy.

A. Mekler (*Monatsschrift für Geburtshülfe und Gynäkologie*, April, 1938) presents an exhaustive survey of the results of the Aschheim-Zondek and Friedman pregnancy diagnosis tests in a series of 1,360 patients. The Aschheim-Zondek test gave a positive result in 96·6% as compared with 93·1% for the Friedman modification, though if doubtful cases are excluded the percentages were 97·8% and 97·4% respectively. In abortion and extrauterine pregnancy the rapidity with which the reaction is lost is in proportion to the intensity of the symptoms. If these develop slowly the reaction is likely to remain positive for a longer period. While the increase in size of the Graafian follicles is not a definite sign of pregnancy according to Aschheim and Zondek, and is noted in non-pregnant women, especially when they are near the menopause or have genital tumours, Mekler considers that it is of value in the diagnosis of ectopic pregnancy if considered in conjunction with the clinical findings. It is also of value in doubtful cases, especially fœtal death *in utero* and missed abortion. This reaction is of use only with the mouse test, and cannot be considered with the rabbit modification. In general, both methods should be employed. If a rapid diagnosis is required the Friedman technique is preferred; but in very early pregnancy the Aschheim-Zondek method gives more certain results.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Section of Oto-Rhino-Laryngology of the New South Wales Branch of the British Medical Association was held on July 27, 1938, at Sydney Hospital.

Suppurative Petrositis.

DR. D. G. CARRUTHERS demonstrated on a specimen Lempert's operation on the cells of the petrous pyramid. Dr. Carruthers then read a paper entitled: "A Review of the Problems of Suppurative Petrositis and of its Surgical Treatment, together with an Outline of the Prophylaxis and Treatment of Otitic Meningitis" (see page 644).

DR. N. H. MEACLE said that he had been struck with the number of children who suffered from acute fulminating meningitis and who on examination were found to have otitis. At the Royal Alexandra Hospital for Children it was recorded that of 76 patients who had died from fulminating meningitis, 58 were found to have *otitis media*. He had operated on a few of these patients when X ray examination of the mastoid process had suggested possible involvement. No evidence of mastoid infection had been found.

DR. ARNOLD BRYANT asked whether the incidence of sixth cranial nerve paralysis with Gradenigo's syndrome could be considered an indication for the Lempert operation.

DR. ERIC P. BLASHKI said that radiographically changes could not be expected to be recognized under a period of three weeks. Then the changes seen would only amount to some decalcification. This had been beautifully shown in a recent number of *Acta Laryngologica*, in an article by Frenckner, of Stockholm. Dr. Blashki could recall two patients who most probably suffered from this disease. Each had had severe pain in the eye. The first had been a young girl with a pneumatic mastoid that had been extensively operated upon. She subsequently developed a sinus thrombosis. She recovered without special attention to her petrous pyramid. The second patient had recently undergone a radical operation. Toward the end of the healing process some fresh discharge from the Eustachian part of the wound occurred, with pain in the eye. After observation for some time it was thought that the X ray picture showed some comparative loss of calcium in the petrous tip; but the condition subsided before anything further was done.

DR. H. S. MARSH could not recall having ever recognized a case of petrositis in his practice. He could remember three or four patients with meningitis who were operated on and who recovered. He thought frequent lumbar puncture a valuable aid to recovery. The last patient that he had in mind, who had recovered, had undergone a Schwarze operation and three lumbar punctures. His recovery took place in spite of the discovery of streptococci in the cerebro-spinal fluid.

DR. A. L. CLOWES was struck with the importance of the pain in the eye, and he agreed with Dr. Carruthers that it was important to eliminate from the diagnosis the possibility of coexisting paranasal sinusitis when patients with mastoiditis seemed unusually ill.

DR. MILTON COUTTS had seen some patients whose condition had been diagnosed as petrositis, but who had recovered after the simple mastoid operation. He was impressed by the demonstration of the operation, but considered it should not be undertaken without very full and serious thought. He agreed that loss of calcification was the only radiographic sign of value.

DR. B. B. BLOMFIELD thought that surgeons should be prepared to meet this condition in order to avoid losing patients. He had seen in the last year three patients suffering from petrositis. The first patient had had a very highly pneumatized bone and had a fistula leading through the posterior semicircular canal. Dr. Blomfield had been able to pass a probe through this to a depth of about 3·75 centimetres (one and a half inches) into a large cavity. It was the occurrence of this case that had led to the present interest at Sydney Hospital in the subject of petrositis. Dr. Blomfield had been unable to find any areas of necrosed bone, but this patient had eventually developed meningitis and died. Dr. Blomfield's second patient, a boy, had had posterior petrositis. He had suffered from double mastoiditis. One side had healed well, but the other had a large mass of postero-lateral cells. These were opened down to the internal auditory meatus and the patient recovered.

Dr. Blomfield said that his third patient, an elderly man, was still under treatment. He had been subjected to an extensive Schwarze operation. A great many very deep retrofacial cells had been found, but recovery had not been as rapid as was usual in these cases and the patient was very ill in spite of treatment with sulphanilamide. He had pain around the eye and ear. X ray films were unsatisfactory, until finally one picture did reveal a difference in appearance between the two sides. There had been little discharge from the wound. About the third week much pus was discharged on one single day. He seemed better after that, and the pain was reduced; but it returned. Lempert's operation was performed. It had proved comparatively easy and had been performed without an anterior incision. Dr. Blomfield had carried the top end of the incision forward and had obtained enough room. There had been no trouble at all with bleeding. The *tensor tympani* muscle had been found to be surprisingly large, as also was its canal. The carotid canal had been uncapped with a gouge and the bone had then been removed with a spoon. No fistula and not much pus had been found, but there had been a considerable amount of cheesy material that had come away with a spoon. The patient at the time of the meeting seemed much better and was free from pain. He was still under medical attention.

Dr. Blomfield held the opinion that when a definite diagnosis had been made the surgeon was in duty bound to clear out the petrous pyramid; otherwise an extradural abscess would probably develop, which was more difficult to deal with. The operation was not an easy one. It was necessary to cut well forward into the zygomatic root and into the squama to get enough room. The operation took him four hours. He began it by exposing as much dura as possible in the middle and posterior fossa areas. He said that Kopetzky had pointed out that meningeal infection arose through infection along the blood vessels running from the bone to the dura.

DR. H. HARRISON said that he had seen only two patients with petrositis. He thought that the operation was a reasonable one. It was probably easier than its description implied, as it seemed that petrositis occurred mainly in cases in which the temporal bone was well pneumatized. Dr. Harrison thought that if conservative surgery did not clear up the disease in a pneumatized tip, Lempert's operation ought to be undertaken.

DR. A. B. K. WATKINS was grateful for the splendid demonstration. He thought that petrositis was definitely recognized as a disease. Kopetzky had described it in 1931. He remembered many patients who had exhibited all the described symptoms and who had recovered without any special surgical procedures. In his opinion there was a tendency for the patients to recover if the mastoid was adequately treated. He found an objection to the use of the term "fistula", since the condition was acute. He preferred this term to be reserved for chronic cases, and thought that in the acute cases the term "tract" should be employed. It had been shown in a number of papers that petrositis could occur in a sclerotic bony pyramid. Dr. Watkins did not think Lempert's operation would be indicated in such a case, as there was not enough space to justify it. The disease was an osteitis, and the opening of some cells in the middle was not sufficient. He had once operated on a patient who had a dead and loose labyrinth. He had expected operation to be easy, but had found that the labyrinth was traversed by the internal carotid artery, which prevented its removal.

Dr. Watkins briefly discussed the operations that stopped short of Lempert's operation. Tracts were sometimes found. Should the patient be suspected of having petrositis, the operation wound should be reopened and tracts should be looked for. The discovery of a tract, if it was followed up, gave a good hope of recovery with a simple operation. If necessary, a radical operation could be performed and a tract searched for in an anterior position, near the Eustachian tube. If a tract was found it should be followed up. If these procedures failed it was a moot point what should be done next. Only 11% of all pyramids were pneumatic, and only the pneumatic ones should be operated on. Dr. Watkins said that he would never undertake to operate on a pyramid unless it was shown by radiography to be a large petrous pyramid and unless in the opposite side well-developed cells were seen at the pyramid tip, since the operation was undoubtedly dangerous. He referred to Dr. Carruthers's statement that the petrous tip cells did not communicate with the mastoid antrum, but directly with the middle ear. In some earlier papers the same statement had been made about the mastoid tip cells; but Dr. Watkins had shown by his own research that it was untrue. He asked for evidence in favour of the present statement. On the subject of meningitis he thought that the results were better according to present accounts, because of earlier recognition and diagnosis. Many of the patients whose condition was now diagnosed used to recover without its being recognized, or with some treatment on medical lines. Dr. Watkins said that he was very keen on insisting on the rule that straightforward, uncomplicated mastoiditis never caused an elevation in temperature to more than 38·4° C. (101° F.). In the initial stage of otitis the temperature might be as high as 39·5° C. (103° F.). With mastoiditis a temperature of more than 38·4° C. (101° F.) meant a complication, usually either a blood infection or meningitis. Lumbar puncture should be performed on the indication of the fever alone. It often gave a clue to a very early meningitis by indicating an increase in the cell count. Many such patients recovered with no further treatment than an adequate simple mastoid operation.

DR. E. MCA. STEEL, from the chair, extended the thanks of the members to Dr. Carruthers for his splendid dissection and for the trouble he had taken in arranging the demonstration and opening the discussion. He said that he could think of some probable cases of petrositis, although he had no personal experience. He recalled in particular one case in which improvement did not occur after a cortical mastoidectomy. A profuse purulent discharge persisted from the aditus and middle-ear regions, and the pulse was persistently rapid. He had performed the radical operation, but the profuse discharge persisted. He had been unable to find a tract, and after a number of weeks the condition cleared up and the wound healed. Dr. Steel considered early lumbar puncture of the greatest importance in meningitis. He remembered a child who had been thought to have a furuncle of the ear and who had a temperature of 38·4° C. (101° F.), but did not appear to be very ill. X ray films of the mastoid at intervals of some days had revealed no abnormality. The patient's condition retrogressed, but lumbar puncture was not carried out. Frank meningitis developed and the mastoid was opened. No abnormality was found near the mastoid antrum, but there was a large extradural abscess in the posterior cranial fossa.

Dr. Carruthers, in reply, said that 95% of the patients who were operated on for mastoiditis recovered. There was, however, a small proportion who died from meningitis. Although labyrinthitis or lateral sinus phlebitis might be the reason for this in a number of cases, petrositis should always be thought of as a condition likely to bring about infection of the meninges. He had been interested to note that each speaker had recalled a case of probable petrositis. Undoubtedly many patients recovered with the ordinary conservative operations. Nevertheless otologists should always be in fear of the development of meningitis, and should be prepared to undertake more radical procedures, such as the Lempert operation, especially in the presence of the symptoms suggestive of deep petrosal infection, such as persistent pain around the eye and a persistent low-grade fever. Also, when the number of cells in the cerebro-spinal fluid was increased, or if the chemical changes he had referred to earlier were detected, further operation was required. He had no doubt that the operation that had been described was a necessary part of the repertoire of a competent aural surgeon. As for patients with supposed meningitis, referred to by speakers, who had recovered without more than routine surgery, Dr. Carruthers remarked that in a number of cases there was a meningeal reaction which was not really meningitis. These patients recovered with only conservative surgery, provided the infecting focus was reached. If a definite diagnosis of petrositis had been made, and the X ray picture demonstrated a cellular pyramid, Lempert's operation should undoubtedly be performed. American writers doubted the occurrence of petrositis in sclerotic pyramids, as had been described by Continental authors. To Dr. Watkins, Dr. Carruthers said that the evidence that petrositis was an extension from the middle ear rather than from the mastoid was that the condition might occur after a radical mastoid operation had been performed. Also, in his study of development of the mastoid and petrosal cells, Wittmaack had shown that it was the epithelium from the middle ear that grew directly into the mastoid and into the petrosal pyramid that was responsible for the process of pneumatization. This developmental process had been shown to extend into the petrous pyramid directly from sites about the Eustachian tube and about the external semicircular canal. He thought that Gradenigo's syndrome was not common in petrositis and was not necessary in its diagnosis. He had seen this syndrome in cases not of petrositis, such as a case of sphenoiditis accompanying mastoiditis. The essential symptoms were the peculiarly localized orbital pain, recurrent or persistent discharge, low-grade fever, and changes demonstrable by radiography.

NOMINATIONS AND ELECTIONS.

THE undermentioned has been elected a member of the Victorian Branch of the British Medical Association:

Harlem, Isaac Cyril, M.B., B.S., 1937 (Univ. Melbourne), 3, Glenhuntly Road, Elsternwick, S.4.

Medical Practice.

STATEMENT BY THE COUNCIL OF THE SOUTH AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION.

WE have received the following communication from the Council of the South Australian Branch of the British Medical Association.

The South Australian Branch of the British Medical Association and the Recent Incest Case.

In view of the desire expressed by some members of the Branch to be informed of the grounds on which the council decided to take no action consequent on a letter received from one of its members with reference to a case of incest, the council is impelled to make the following statement.

1. The council realizes that public opinion is immensely and medical opinion is much in favour of the legal termination of pregnancy in cases of rape and incest. This law obtains in Denmark. The council, however, has not yet considered what action, if any, it should take in respect of alteration of the law concerning abortion. Before any action is taken the members of the Branch will be consulted. The existing law in South Australia, and it alone [Section 81(b), *Criminal Law Consolidation Act*] governs the procuring of abortion. In the light of that

law the council has had no difficulty in formulating its views. The law is as follows:

> Any person, who, with intent to procure the miscarriage of any woman, whether she be or be not with child, unlawfully administers to her, or causes to be taken by her, any poison or other noxious thing, or unlawfully uses any instrument or other means whatsoever with the like intent shall be guilty of felony, and liable to imprisonment for life.

The use of the word "unlawfully" implies that Parliament had in mind that such an act might be done "lawfully" if it were necessary to preserve the life of the expectant mother, and this in practice means her health and sanity; for without either life is worthless. The judge in the Bourne case, in addressing the jury, said that it was obvious that the act if done by skilled persons without risk to the patient and on purpose to save the life of the mother must be lawful. The law did not permit the termination of pregnancy except for the purpose of preserving the life of the mother; but those words ought to be construed in a reasonable sense. If the doctor was of the opinion on reasonable grounds and on adequate knowledge that the continuance of pregnancy would probably make the woman a physical wreck or a mental wreck, the act was lawful. No doubt doctors in performing abortion would act only in consultation with some other doctor of high standing.

To this judicial ruling the council desires to add that the law does not permit the termination of pregnancy for humanitarian or eugenic reasons.

In a letter by Charles A. H. Franklyn, M.D., M.R.C.S., published in *The British Medical Journal*, August 13, 1938, page 373, it is stated:

> However, it is not for us medical men to make or to modify the laws of the land (apart from pressing for necessary reforms as citizens), but, being in a special degree of relationship and trust with the State, to observe the laws as we find them to-day.

2. At no time has the council been approached by the Government or Crown law authorities with reference to the recent case of incest. The circumstances under which the case was brought to the notice of the council were as follows. After giving evidence in the police court that the girl was twenty-six weeks pregnant, the medical practitioner, who had been subpœnaed, requested the magistrate to receive from him a recommendation to be forwarded to the proper authority. The recommendation was couched in words like the following:

> In consideration of the very grave doubt as to the future mentality of the child to be born and the danger to the future health and mental state of the child mother, I beg to recommend that doctors learned in such matters be asked to confer with a view to ensuring the non-survival of the infant.

The practitioner concerned subsequently wrote to the President of the South Australian Branch stating:

> . . . it is in my opinion an occasion warranting the consideration of the council. I am, alas, not an authority, but I do deem this an instance when, if recognized expert opinion holds that pregnancy should be terminated, such action could be pursued.

The council emphatically asserts that it is not its province officially to advise members of the Branch on the professional care of individual patients. Within the ranks of the South Australian Branch of the British Medical Association are specialists in all branches of medicine, fully competent and willing to give advice, if sought, on every aspect of a patient's health. It is germane to this question to remember that in the Bourne case, action with a view to the termination of the pregnancy of a girl of fourteen, who had been raped, was initiated by a lady organizer of the Schools Care Committee, who consulted Dr. Joan Malleson. She in turn consulted Dr. Alec Bourne. At no time prior to the bringing of this incest case to the notice of the council had the members of the council or any other doctor been consulted by the girl's mother or by anyone acting on behalf of the mother, with reference to the girl's health, physical or mental. In the course of the discussion which followed on the reading of the medical practitioner's letter and his statement made at the time, it was pointed out to him how the opinion of specialists might be obtained to determine whether the birth of the girl's child was fraught with danger to the life of the mother. The council deemed no action other than this to be necessary.

Post-Graduate Work.

COURSE IN OPHTHALMOLOGY AT SYDNEY.

The New South Wales Post-Graduate Committee in Medicine announces that, in conjunction with the Ophthalmological Society of New South Wales, it will hold a course in ophthalmology from Monday, October 31, to Friday, November 11, 1938. The programme is as follows:

Monday, October 31.

At the Royal Prince Alfred Hospital:

9.30 a.m.—Registration.

10 a.m.—Dr. H. L. St. Vincent Welch: Methods of examination of the eye; demonstration of apparatus and instruments used.

At the Eye Department, Sydney Hospital:

2 p.m.—Out-patient attendance; clinical demonstration.

Tuesday, November 1.

At the Eye Department, Sydney Hospital:

9.30 a.m.—Dr. C. K. Cohen: "The 'Red' Eye (Superficial Affections), Acute Conjunctivitis, *Ophthalmia Neonatorum*, Trachoma, Corneal Ulceration, Interstitial Keratitis *et cetera*".

2 p.m.—Out-patient attendance; clinical demonstration.

Wednesday, November 2.

At the Royal Alexandra Hospital for Children:

10 a.m.—Dr. N. M. Gregg: "Squint".

(Afternoon free.)

Thursday, November 3.

At the Royal Prince Alfred Hospital:

10 a.m.—Dr. James Flynn: "Iritis and Glaucoma".

At the Eye Department, Sydney Hospital:

2 p.m.—Out-patient attendance; clinical demonstration.

Friday, November 4.

At the Eye Department, Sydney Hospital:

9.30 a.m.—Dr. R. B. North: "Cataract".

11 a.m.—Dr. C. G. H. Blakemore: "Proptosis".

At the Out-Patient Department, Royal Prince Alfred Hospital:

2 p.m.—Out-patient attendance; clinical demonstration.

Monday, November 7.

At the Royal Prince Alfred Hospital:

10 a.m.—Dr. E. A. Brierley: "Headache".

At the Eye Department, Sydney Hospital:

2 p.m.—Out-patient attendance; clinical demonstration.

Tuesday, November 8.

At the Royal Prince Alfred Hospital:

10 a.m.—Dr. G. Waddy: "Optic Disk and Neurology".

At the Out-Patient Department, Royal Prince Alfred Hospital:

2 p.m.—Out-patient attendance; clinical demonstration.

Wednesday, November 9.

At the Royal Prince Alfred Hospital:

10 a.m.—Dr. H. de Burgh: "Commoner Fundal Conditions".

(Afternoon free.)

Thursday, November 10.

At the Royal Prince Alfred Hospital:

10 a.m.—Dr. Colin Ross: "The Eye in Relation to General Diseases".

At the Out-Patient Department, Royal Alexandra Hospital for Children:

2 p.m.—Out-patient attendance. (Special reference to the commoner eye diseases of infants and children.)

Friday, November 11.

At the Royal Alexandra Hospital for Children:

10 a.m.—Dr. W. M. C. MacDonald: "Injuries to the Eye".

At the Out-Patient Department, Royal Prince Alfred Hospital:

2 p.m.—Out-patient attendance; clinical demonstration.

The number attending this course will be limited to fifteen. Registration will be made in order of application until fifteen are received. The fee for the course will be five guineas. Application should be made (with cheque enclosed) to the Secretary, New South Wales Post-Graduate Committee in Medicine, the University of Sydney.

The course will not be held unless at least six applications for registration are received.

Obituary.

ERNEST SANDFORD JACKSON.

Ernest Sandford Jackson, who died at Brisbane on June 29, 1938, was the son of John Henry Jackson, who owned Sandford station, near Sandford, Victoria. His great-uncles, William and Samuel Jackson, were among the earliest settlers in Victoria; in fact, they were with Faulkner's party when the first hut was built on the site of the future city of Melbourne.

Jackson was born in Victoria on July 18, 1860. He was educated at Geelong Grammar School and the University of Melbourne. He entered Trinity College, in the university, at the age of fifteen years, graduating in medicine with honours in 1881. Actually he passed his final examinations before he had reached his majority. Among sporting activities at school and college he had the greatest love for rowing, at which he attained considerable prowess. He retained his fondness of games throughout his long active life. He was a keen angler and an excellent horseman.

In 1882 he became a resident medical officer at the Melbourne Hospital, and later in the same year he went on to Brisbane, where he was appointed medical superintendent of the General Hospital. He retained this position until 1898, when he commenced practice at Brisbane and was appointed honorary surgeon to the hospital. On his retirement, after many years of service on the active staff, he was made an honorary consulting surgeon.

In 1885, while still medical superintendent, he founded the Brisbane General Hospital training school for nurses, the first to be established in Queensland. This was a work that any man could contemplate with pride.

In the Great War he served in Egypt as a major in the Australian Army Medical Corps during 1914-1915. He was invalided home in 1916 and served as a lieutenant-colonel on home service for the remainder of the War.

His professional reputation was high, on account of both his skill as a surgeon and his lofty ideals. He had a very large surgical practice and was known and honoured by people in every part of the State of Queensland and by his colleagues throughout Australia. He retired from active practice in 1934, and thereafter devoted a great deal of his time to gardening at his home at Victoria Point.

Jackson was a foundation member of the Queensland Branch of the British Medical Association; he was President of the Branch in 1895, 1911 and 1926, and was a member of the council for varied periods until 1929. He was President of the Queensland Medical Board for a number of years. He was a member of the Royal Commission on Hospitals in Queensland in 1930. Apart from these public-spirited activities and his high professional attainments he will probably best be remembered by his colleagues as an historian. Particularly during his later years he took a great interest in the history of his native country. He was the author of a number of papers on the subject, and was regarded as an authority.

In 1931 the Queensland Branch honoured him by inaugurating a lecture known as the Jackson Lecture, to be delivered annually, "to place on permanent record the appreciation of the Branch of the distinguished services rendered by Dr. E. Sandford Jackson over an extended period of time". In view of his keen interest in medical history, it was decided that the lecture each year should be concerned with this subject. At the invitation of the Council of the Queensland Branch, Jackson himself delivered the first lecture, on September 4, 1931, his subject being "Some Voyages Connected with the Discovery of Australia: Their Medical History".

Jackson was one of the pioneers. Among the names of those who blazed the trail his will have an honoured place.

The sympathy of the medical profession in Australia is extended to Mrs. Jackson and her family in their bereavement.

Dr. A. C. F. Halford writes:

When I joined the staff of the Brisbane Hospital in 1895, E. S. Jackson was the medical superintendent and J. O'Neil Mayne was my co-resident. Brisbane was then a backward city, with barely 100,000 people; the roads, over which lumbering buses rolled, were bad. Yet I found a most efficient hospital, staffed by nurses whose skill, devotion and wide knowledge of theoretical and practical nursing astounded me. As a young graduate I did not hesitate to take advantage of acquiring training from these experienced nurses, Miss Marks, the matron, Miss Chatfield, her chief of staff, and the senior ward sisters. Francis Hare had instituted the cold bath treatment of typhoid fever with the thorough cooperation of the nursing staff, as described in his book. Jackson was teaching the advantages of open-air treatment of all chronic infections, and generally keeping all hands busy. He was almost a martinet and without compromise; his slogan was, "The patient is always right". The comfort and safety of the patient were never to be in jeopardy, and any dereliction of duty met with severe reprimand and not uncommonly dismissal. In his own sphere he never spared himself. Complaints rarely got past him. Even the honorary staff felt they had to watch their steps. Next door, at the Hospital for Sick Children, Jefferis Turner was winning marvellous victories with large doses of serum in diphtheria, and was skilfully using and teaching the staff in emergencies to use tracheal intubation. If ever team work had a birth-place it was at these two institutions. Naturally I was happy and voted myself lucky to find pleasant associations in which to gain valuable experience. In particular, the fever wards, generally full of enteric patients, were most instructive. With the legacy Hare left, Jackson had his staff alert to recognize quickly the changes in the condition of patients due to any of the many complications of the disease. The charge nurse or the ward nurse knew exactly what to do and what to prepare for the resident, who was urgently summoned. Without being invidious, for all were excellent, the charge nurse was always so polite, almost obsequious, with her "yes, doctor" punctuating every few words Jackson said to her, but he could not, however obviously irritable it made him, disturb her polite and imperturbable calm. She and ward

nurse Capner were typical examples. The former married a doctor and settled down in New Zealand, and the latter became well known as matron for many years of the Carlton Women's Hospital. She stayed on duty continuously for three days while one of her typhoid patients in the same period lost seventeen pints of blood, as shown in the hospital chart still in my possession. He was 153 days in hospital before recovering, after many relapses. Hare had not then discovered and recommended amyl nitrite as a valuable hæmostatic in all cases of internal hæmorrhage. This use of the drug, a sheet anchor, seems to have been forgotten, though very helpful in hæmoptysis in particular. Jackson has told the readers of the journal the history of the Brisbane Hospital from its foundation in his historical addresses. It now remains for others to speak of his part in bringing it to the high state of efficiency to which it rapidly attained.

Miss Weedon left London and arrived in Brisbane in October, 1885, at the suggestion of a friend, who said she understood that there were no trained nurses in Queensland. She was introduced to the late Mr. John Petrie, who was chairman of the hospital committee. Miss Weedon's credentials as a graduate of Saint John's Hospital and Sisterhood and the Chelsea Maternity Hospital, and also the Charing Cross Hospital, so impressed Mr. Petrie that he at once asked her to take over the training of the nurses, and she began her duties a month later. This position she held for five years, and I am happy to say she is still living amongst us in cheerful retirement. In the historical references at my disposal there is some confusion of dates. In the well-compiled and interesting book "A General History of Nursing", by Lucy Ridgeley Seymer (Faber and Faber, Limited, 1932), it is stated that the training school in Brisbane started in 1888, which is obviously an error. Miss Crosse succeeded Miss Weedon; then Miss Marks was appointed, and was matron during my residency. She informs me that she joined the staff in May, 1887, and that the practical training was augmented by lectures a month later. These were presumably given by Miss Weedon and Dr. Francis Hare, who acted as medical superintendent during Jackson's absence in England in 1888.

Jackson was then twenty-seven years of age, having qualified when he was in his twentieth year. He told me that when first appointed as a resident surgeon in 1881 he could not sign death certificates or receive his registered degrees because he had not reached his majority.

On his return from England this young man threw all his energy and organizing ability into the final shaping of his training school, claiming it to be the first founded in Australia. That Jackson was a reformer, and a bold one at that, is well illustrated by the edict he issued about the year 1889, that skirts of uniforms were to be twelve inches from the ground. The shrill protesting outcry from the nurses at having to expose so much leg was unheeded, and those who let a hem down did not escape the medical superintendent's eagle eye. They were mollified when they saw that the matron had to cut off her train. Miss Florence Chatfield, O.B.E., in a most interesting lecture to Queensland nurses, published in *The Australian Nurse's Journal*, April, 1936, describes Miss Weedon thus:

> It can be truly said of Miss Weedon that she was Queensland's "Lady with the Lamp". Possessed of outstanding ability and moral integrity, she was a good disciplinarian and an excellent teacher who upheld all the best traditions of nursing and set a standard of a very high order.

The first qualifying examination for nurses was held in 1890. From then on came annually the well-trained and capable nurses who staffed the hospital and served the community in town and country so ably, as I was able on coming to Brisbane to judge, and later to choose one to be my loving wife. Jackson had already set this example; the sweet unassuming devotion to her family, to nurses and to such institutions as the Bush Nursing Association has drawn the deepest sympathy towards Mrs. Jackson in her widowhood.

Since writing the above I have learned that E. S. Jackson has left behind him a book of the nature of an autobiography. He postponed its publication on account of the estimated cost, about £300. In my remarks I have endeavoured to check dates and facts as carefully as possible, and I hope they will find confirmation in the story Jackson has told of what he could rightly deem the most important of his life's work, and one to be judged as most valuable. Surely there can be found among the friends and admirers of Jackson a sufficient number to subscribe for the publication by permission of a limited number of volumes, and I confidently leave the suggestion in the hands of the historical section of the Queensland Branch of the British Medical Association to do this service to his memory and to medical history.

Miss Florence Chatfield writes:

My association with the late Dr. E. Sandford Jackson during his superintendentship of the Brisbane Hospital dates from the year 1889, when I entered that institution as a probationer and continued until, as deputy-matron, I retired from the staff in 1898. Thus throughout my early nursing years I had every opportunity to judge of Dr. Jackson's character and personality, and observe his ability as administrator of a large institution.

These were, in surgery, transitional years, and ranged from the days when surgeons performed operations clothed in their everyday apparel to those when they were arrayed in the white armour of the present-day theatre.

When I joined the hospital in 1889 Miss Weedon was matron. This lady, who was a graduate of Saint John's Hospital and Sisterhood, Charing Cross Hospital, and Chelsea Maternity Hospital, had been appointed matron of Brisbane Hospital in 1885, and with Dr. Jackson established the first nurses' training school in Queensland. With their most able direction and management these foundations were well and truly laid, and the debt that Queensland nurses owe to these pioneers is incalculable. Under their training the tenets of the Florence Nightingale pledge for nurses were thoroughly instilled, and trainees were constantly reminded that the welfare of the patient and loyalty to those in command should be their first consideration.

Dr. Jackson had many fine qualities, but outstanding was the fact that he was a man of honour. Of him delinquents walked in dread, but those who conscientiously sought to do their duty had no cause for fear.

The evolution in hospital technique at this time called for many new rules and regulations, and at times these met with criticism and even opposition; but it was soon learned by the staff that an order from Dr. Jackson had to be unquestionably obeyed. One order, which he has referred to as "the battle of the skirts", that the nurses' uniforms must be a regulation number of inches from the ground, met with strong opposition; but his word was law, and soon the then unfashionably shorter skirts were accepted.

Dr. Jackson showed unerring judgement in selecting heads for his various sub-departments. Of those selected he expected much, and through the trust and confidence he placed in them he received much. The wisdom of his judgement was proved by the excellence of the results achieved, and by the fact that so many officers selected by him grew old in giving faithful and efficient service to the institution.

By the staff he was regarded as a strict disciplinarian, but to the nurse brought before him for a mistake he showed, time and again, that he could temper justice with mercy. For the one who wilfully deceived, punishment was swift and sharp.

Dr. Jackson was one who at no time spared himself in ministering to sick and suffering humanity. As one intimately associated with him in both his hospital and private work I can state that it would be impossible to estimate the tremendous amount of gratuitous service he rendered to those in need.

Now, after a lapse of forty years, many nurses still recall their days in Brisbane Hospital and the respect and admiration they had for their medical superintendent.

All feel that a great man has passed.

Proceedings of the Royal Commission Appointed to Inquire into Matters Pertaining to National Health Insurance.

Friday, September 30, 1938.

At the opening of proceedings on September 30, 1938, Mr. Gain read a letter that Dr. A. J. Parer had written to Mr. Abrahams. This was as follows:

Maleny, Queensland,
Sept. 26th, 1938.

Mr. Abrahams, K.C., Sydney.

Dear Sir,

When giving evidence before the Royal Commission in Brisbane I expressly asked, and permission was given that there would be nothing published that would identify the practices in London with which I was associated.

In the last issue of THE MEDICAL JOURNAL OF AUSTRALIA this undertaking has been broken, and I would like you to draw official notice thereto. I refer to the issue of 24th inst. on pages 529-530.

This would be objectionable enough if the report were an accurate record but its inaccuracy makes it intolerably unjust to the men who conducted the Wandsworth practice.[1]

This practice was a good class of practice as panel practices go in London, and was the best of which I had experience.

It was in the other areas that the very poor type was conducted.

The men running the Wandsworth practice were earnest, keen and competent. It was no fault of theirs that the large number of services required under the system precluded them giving a medical service that they would like to have given.

When I referred to the standard of service being about equal to that given by a railway or other man trained in first aid I was referring to the other practices in the poorer quarters of London. These first aid men do a lot of medical work as honorary ambulance men in Queensland in areas far removed from medical aid. They treat minor ailments, provide dressings, ointments and medicines. Some of them even suture wounds and attend women in cases of miscarriage or confinements preparatory to sending them to the doctor or hospital. This is even more than that given at some of the medical practices in the congested industrial areas of London, where there would be no suture equipment to use if the *locum* did desire to use it.

I wish you to bring this letter to the notice of the Commission, and, if possible, to have it read in open Court and given publicity so that the injustice done to these Wandsworth practitioners will be mitigated.

Mr. Gain: Dr. Parer has enclosed the report that is contained in THE MEDICAL JOURNAL OF AUSTRALIA. I have not had an opportunity of checking it with Dr. Parer's evidence.

Dr. Hartley Wilson Austin, Medical Superintendent of Sydney Hospital, was called as a witness by Mr. Dovey and gave detailed evidence and figures concerning the administration of that hospital, dealing particularly with its capacity, the casualty department and the out-patients' department.

Mr. Dovey: So each new patient [in the casualty department], of course, would take up some little time on the part of the doctor?

A.: Yes, depending on the nature of the case. One case may take only two minutes, and another fifteen or twenty minutes before he could be released.

Q.: On the average, how many would he get through in the casualty department in an hour, taking, say, the first 50 that are lined up in the morning?

A.: You would not do more than 25 to 30 as the maximum, that is, of ordinary cases. Twenty-five to each man would be the absolute maximum.

The Chairman: Obviously, you have rush or peak periods?

A.: Yes.

Q.: Therefore you cannot take the average over the whole period as a good criterion. At the same time, you cannot take the rush at the peak period; that is to say, the number of patients a man gets rid of at the peak period cannot be taken as the normal practice?

A.: No.

Q.: How many do you say would be the utmost a man could get through in an hour?

A.: Approximately 25, and that would exclude any accident cases. One accident case may take a doctor 25 minutes or 30 minutes by itself.

The Witness (continuing): People coming in with coughs and colds or something like that could be seen possibly at the rate of 25 in an hour, and drafted to the out-patients' department, or given advice as to treatment.

Mr. Abrahams: Our only contention in regard to this aspect of the matter is that we should try to take the figures all the year round as to how many can be treated in a certain time.

The Chairman: The point is that a panel doctor will have certain hours set aside for panel work—something in the nature of lodge hours. That may be, say, two hours in the morning and two hours in the afternoon. With the panel work the people would come during that allotted two or three hours, but I understand that in the hospitals the out-patients may come during a period of something like nine hours?

A.: They can come at any time.

Q.: But I think you said that the recognized day was nine hours?

A.: Yes.

Q.: If they came in an even flow during those nine hours we might be able to compare the panel working period with the hospital working period and take the same average. But that does not appear to be possible in these circumstances, because it appears that in the nine hours you sometimes have a lull?

A.: Yes, quite often.

Q.: During the lulls the medical men are waiting about?

A.: Yes. It is impossible to compare hospital practice with general practice, because such cases as can be sent to the out-patients' department need no further examination. In the case of a number of the patients you have only just to look at them. They may have a lump or a rash, and you just refer them to the out-patients' department.

Dr. John William McNamara, the Medical Superintendent of Saint Vincent's Hospital, gave evidence concerning the same matters that Dr. Austin had dealt with, as also did Alexander Skeffington Johnson, the Deputy Superintendent of the Royal Prince Alfred Hospital.

Mr. Dovey: That is all the evidence I propose to call in Sydney. Of course, on both sides there will be a summarizing of the figures by the accountant and the actuarial evidence.

The Chairman: I anticipate that will be tendered here.

Mr. Dovey: I think that would suit the convenience of everybody.

The Chairman: I think so.

At 12.5 p.m. the Commission adjourned until 10.30 a.m. on Tuesday, October 4, 1938, at the Commonwealth Arbitration Court, Melbourne.

[1] The report of Dr. Parer's evidence in the issue of September 24, 1938, is accurate, according to the official transcript. There is no mention in the transcript of any assurance that any part of Dr. Parer's evidence would not be reported, nor did this journal give any such assurance.—EDITOR.

Tuesday, October 4, 1938.

The sittings of the Commission in Melbourne were commenced on Tuesday, October 4, 1938.

Mr. Dovey: If the Commission pleases, by arrangements made with my learned friends Mr. Abrahams and Mr. Slater, who is appearing for the friendly societies in Victoria, and subject to the permission from the Commission, I propose that this present portion of the Melbourne sittings, which will occupy only this week, will be conducted in the following manner. Witnesses will be called by my learned friend Mr. Abrahams, representing the British Medical Association, and I am informed by Mr. Slater that, at some time or other, but not this week, evidence will be put before the Commission by him on behalf of the friendly societies of Victoria. I do not propose at this stage to indicate the nature of that evidence. Mr. Slater will do that. If the Commission will pardon me, I would like publicly, and through the Press, to direct the attention of the public to the importance of the sittings of this Commission in Victoria. Its importance is twofold. First of all, we appreciate that Victoria, by reason of its population, is one of the States in the Commonwealth from which it might reasonably be expected that we shall obtain valuable evidence upon matters which call for inquiry. Secondly, these sittings are of importance because it will be remembered that, during the negotiations between the representatives of the British Medical Association and the official representatives of the Government in March and thereafter of this year, it was urged by the representatives of the medical profession that the friendly society rate which obtained in this State, namely, the rate of 20s. per member, was a rate which had been imposed on the doctors and accepted by them under duress. It will also be remembered that that statement, without necessarily being accepted fully by the representatives of the Government, was accepted to this extent, that it was agreed between the parties to the negotiations that, for the purpose of weighing the *per capita* rate, or the friendly society contract practice, Victoria should be regarded on the same footing as New South Wales. That is to say, Victoria was taken in at 26s. instead of at 20s. The British Medical Association and the friendly societies are represented here, and it will be interesting to see whether or not the contentions of the medical representatives in Melbourne and Sydney in the early part of this year were justified. I do not propose to say more than that at this stage.

After discussion as to whether the Victorian friendly society rate of 20s. should be taken into consideration or not, Mr. Abrahams stated: I should like to say this, that there seems to be an idea on the part of the Crown that when anything is said by the medical profession it binds the medical profession for all time; when anything is done by the medical profession, it binds the medical profession for all time; but when anything is done by the National Health Insurance Commission, they have only to go back on it and a new set of circumstances arises. Now the National Insurance Commission was prepared to accept the statement that the Victorian rate was too low. If that is so they are just as bound by that acceptance as the doctors are. If Mr. Brigden goes back into the matter I have no doubt that he will consider it in accordance with his duty to accept the statement as fair; otherwise there would be the possibility of giving away public money. If, as in Sydney and in Brisbane, the friendly societies have supported the doctors' case, there is no reason why he should try to create a breach between the friendly societies and the doctors in Melbourne.

Mr. Slater: There will be no such breach as far as we are concerned.

Mr. Abrahams: All my submission on that matter will be that the National Health Insurance Commission has agreed that it is right to consider the Victorian rates too low, having regard to all the relevant circumstances.

Mr. Dovey: It is not binding.

Mr. Abrahams: But it is a circumstance that must be taken into consideration. One cannot be bound by past conduct without the other being bound by past conduct. It has been suggested that because the doctors have done a certain amount of what is regarded as concessional work, they have been satisfied to do concessional work. That is perfectly true, and it is a circumstance to be taken into consideration; but it is also a circumstance to be taken into consideration that the National Health Insurance Commission does take up a part of that type of work in Victoria. The Victorian rate, when one regards the rest of Australia, is a low rate.

Mr. Abrahams stated that the results of the census taken for Victoria by the British Medical Association in conjunction with the Government Statist as to the unit represented by a friendly society member would be placed before the Commission.

Mr. Dovey expressed the hope that he would be able to put before the Commission the results of the inquiries on this point made by the friendly societies which were in the process of compilation by the National Health Insurance Commission.

Dr. Eric Leonard Cooper was examined by Mr. Gain. He stated that he was at present carrying on a consultant practice in Collins Street, Melbourne, and that during the years 1932 and 1933 he had had experience of panel practice in England.

Dr. Cooper gave a detailed description of his experience and impressions of panel practice in England, and was cross-examined thereon by Mr. Dovey. He was also questioned concerning his experience as a resident medical officer at the Royal Melbourne Hospital.

Mr. Gain: Mr. Dovey asked you about examining patients. Supposing that a patient came to you for the first time and complained that he had a slight cough and was feeling a little run down, how long do you think it would be necessary to take over that case?

A.: In my present practice at the present time I would not take less than three-quarters of an hour. The nurse in my rooms is instructed to allow three-quarters of an hour for any patient coming along for examination.

Q.: You are a tutor and lecturer now?

A.: Yes.

Q.: I am referring to the case of the general practitioner. Take the general practitioner carrying on in practice, what time do you think he should allow over such a case?

A.: Do you mean including the taking of the history of the patient and examination of the patient? If so, I would say that he could not do it under fifteen minutes. In fact, he ought to take anything up to twenty or twenty-five minutes. In that case you mentioned, of a man with a cough and feeling run down, it may be that he would have symptoms of tuberculosis.

Q.: Take the case of an ordinary patient coming along with a headache and saying that he felt run down; how long do you think that a general practitioner could properly take over such a case?

A.: I would say a minimum of a quarter of an hour to make a detailed history and examine the patient.

Mr. Dovey: As regards that three-quarters of an hour you spoke about, you would charge a specialist's fee for such an examination?

A.: Yes.

The Chairman: Specialists charge a special fee because they give special attention, special time and special skill.

Mr. Dovey: Yes, to special people.

The Chairman: We have to deal with the ordinary average practice, somewhat similar to that which is given by a lodge doctor to his patients. That is what we have to do. We must not break in on any kind of practice which is special in its nature and dissimilar from the ordinary practice.

Dr. Robert Mervyn Shaw, of Mordialloc, was examined by Mr. Abrahams.

Mr. Abrahams: I would like to intimate that the Commission has more than once expressed the view that there are certain matters which it is undesirable to labour, such as, for instance, night fees and a whole lot of things of that sort. I recall that the Commission expressed that view in Queensland. Therefore, with the approval of the

Commission, what I am proposing to do now is to deal with those matters only with a few witnesses in each State, merely to show that the practice does not appear to differ as between States, and then to drop it, as has been done in the other States. For example, the course at the university, the matter of buying a practice, the night fees, and so on. I shall run quickly through those matters merely to indicate that the course of practice is the same in each State, and then, when I come to subsequent witnesses, I shall no longer ask them such questions. If my friend wishes to challenge them in any way, I will leave it to him to do.

Dr. Shaw stated that he was the medical officer of eighteen branches of twelve friendly societies. He described the nature of his lodge practice and patients.

The Chairman: What class of people have you consulted as to their ability to carry on the lodge payments in future as well as the insurance payments?

A.: Just the average type of lodge patient, who is almost invariably an ordinary wage-earner.

Q.: Have you any idea what would be the amount of wage that they would earn?

A.: Approximately the basic wage or a little over.

Mr. Abrahams: Will you please try and think back and see if you can recall some of the individuals who have mentioned this to you?

A.: There is one in particular; he is a local gardener who is kept pretty well fully employed. I should say that he keeps occupied pretty well all the year round, and he told me quite definitely that he had great difficulty in meeting his ordinary lodge subscription, and that if he had to transfer that subscription to the national insurance and pay any more to the lodge he did not think that he could do it. He was indeed very worried about his wife and the kiddies—how they would be provided for.

Q.: You mean to transfer to the national health and to pay more to the lodge? The position is a little confusing?

A.: He pays approximately 1s. 6d. to the lodge now. He would have to transfer that to the national insurance, and he said that he thought he would be unable to make any contribution to the lodge to provide for his wife and children.

Remuneration under the *Workers' Compensation Act* and fees for confinements were then discussed.

Mr. Abrahams: Have you had any difficulty in getting patients into the hospital?

A.: Right into Melbourne, the distance is about fifteen miles.

Q.: Have you had any recent experience?

A.: Quite recently I had a case of pneumonia. The husband was unemployed, and the house was quite a small unsuitable place to nurse a pneumonia, and I rang up every hospital in the city on one day, and again on the second day, and again on the fourth day in one week, and on each occasion I was unable to get a bed, and finally I had to get her nursed at a neighbouring institution—I had to get her nursed by a neighbour instead of getting her into hospital at all.

Q.: Was your patient very sick?

A.: Yes, I was quite worried about her.

Q.: Has that been your experience on other occasions?

A.: I have invariably had difficulty. It is quite a nightmare to get your patients into a public hospital.

Dr. Shaw gave the details of the special lodge investigation which he, amongst others, had been asked to make, and was questioned thereon and concerning his records.

Mr. Slater, who appeared for the Friendly Societies' Association of Victoria, examined Dr. Shaw about the Wasley award and relations between the British Medical Association and the friendly societies, and then Mr. Dovey examined him concerning his answers and records relative to the *questionnaire* that had been sent to him.

Dr. Shirley Elliston Francis, of Belgrave, on the Dandenong Ranges, about twenty-five miles from Melbourne, was examined by Mr. Gain and Mr. Abrahams. Dr. Francis described it as a holiday resort and a district of small farms. He was questioned by Mr. Gain concerning mileage.

Mr. Gain: Your district is a somewhat unusual one?

A.: It certainly is.

Q.: Tell the Commission why the normal position does not obtain in your district as regards the mileage?

A.: Owing to the fact that it is a very hilly district, and the mountain ranges run in some places parallel to each other. The only way of getting to various spots is by circuitous routes, which run zig-zag, and up and down, and through steep hills, or around them, or along valleys.

After giving details concerning difficulties of transport and difficulties which he experienced in getting urgently sick people into hospitals, Dr. Francis was examined concerning his practice generally. He mentioned the population of visitors during the summer months and discussed the loss of income received from them that would occur under national insurance. He was then asked by Mr. Abrahams: You kept a record of how many of those 94 patients would come under the national health insurance scheme. Can you tell me how many of them there were?

A.: As far as I can ascertain, there would be 46.

Mr. Gain: You have been asked to consider the question of the value per service of included as against excluded services, is not that so?

A.: Yes. On the figures worked out yesterday, the excluded figures came to slightly less than the included figures.

Dr. Francis was later cross-examined by Mr. Dovey on this point.

Mr. Dovey: You had never considered this question of included and excluded services before yesterday, had you?

A.: How do you mean?

Q.: The relative value?

A.: No.

Q.: I suppose you had always been under the impression, rightly or wrongly, that the sort of work like operations for tonsils, anæsthetics and things of that sort, and confinements, for the time you devoted to your work you were better paid for that than the ordinary physician's service? That was the impression you were under yesterday?

A.: With the exception of the maternity cases, yes.

Q.: You came to the conclusion you were better paid for them?

A.: Yes.

Q.: . . . You are still of the opinion that for the time you were engaged in giving these excluded services you were better paid than for the included services?

A.: No.

Q.: You did not see any difference? But prior to yesterday you were of the opinion that at per service the excluded paid better than the included?

A.: Yes, apart from maternity.

Q.: Including maternity with all the others, did you have any views on the matter before yesterday?

A.: I did not devote a great deal of thought to it apart from maternity. In regard to the time I naturally thought the excluded services would be slightly better.

Q.: What caused you to change your mind?

A.: The fact that the accountant checked over my books yesterday afternoon and my figures there. He asked me for certain figures which I was able to give him, and he worked it out and told me that the excluded services were definitely less remunerative on my figures than the included.

Q.: So that when you said just now that in your opinion included services were of less value, it was not your opinion at all, but the opinion of an accountant who looked at your books yesterday?

A.: Yes, and if the books are correct.

Q.: But if he told you so you believed him?

A.: Naturally; I checked the figures over.

After further cross-examination by Mr. Dovey and reexamination by Mr. Abrahams, particularly as to Repatriation Department work, the witness withdrew.

Dr. Montague Owen Kent Hughes, of Moonee Ponds, which he described as "semi-industrial—more industrial than otherwise", was examined by Mr. Abrahams. He was

examined first upon his answers to the "yellow form" *questionnaire*. He stated that each member of a friendly society on his list represented 2·2 persons.

He estimated 30% as the figure of diminution he expected in his practice as a result of the national health insurance scheme. He was examined in some detail concerning this by Mr. Abrahams, who stated that it was "rather high as compared with what we have had anywhere else".

Mr. Abrahams: You have checked it over and you cannot make it any different; it is 45%?

A.: That is so.

Q.: Then you reduced it to 30%—on what basis?

A.: On the basis of the lodge figures. The number of excluded services and the number of included services.

Q.: You found, I think, that proportion of the lodge extras as far as service was concerned was about one-third of your total lodge service, and so you have reduced the 45 by one-third and brought it down to 30?

A.: Yes.

Dr. Kent Hughes was next examined as to his lodge practice.

Mr. Abrahams: Do you find there is any tendency for lodge patients to consult you without real necessity?

A.: Yes. They come up for minor illnesses, such as colds, that normally private patients do not consult about.

Q.: In certain cases they have come to get their medicine. Outside those cases do you think they consult you more than private patients do?

A.: Quite a lot.

Q.: I suppose mileage is not a very important matter with you?

A.: It is not important.

Q.: Do you ever charge it?

A.: Yes, occasionally.

Q.: And you find that it stops them from calling you out?

A.: On a couple of occasions I did not charge it, but it became necessary to do so.

Mr. Dovey: I gathered that the lodge patients are more trouble than private patients?

A.: Yes.

Q.: From your records, you receive considerably less from the lodge patients than from private patients?

A.: Yes.

Mr. Dovey: Your Honour remembers something that was said this morning. I will make the position quite plain. One sees here a doctor with a very large private practice, who says that the lodge patients are more trouble to him than the private patients. On his figures he receives very little from the service he renders, and yet we find he has rejected more than two a year for the last five years. It would average round about 5%. He has, despite all these disadvantages, taken on to his list a considerable number of new members.

The Chairman: Of course, that is the object of your attack. Could you not have put it in two or three words: "How many additions have you made to your lodge patients in the last five years?"—seventy, he says.

Dr. Kent Hughes was then examined by Mr. Slater.

Mr. Slater: Do you not agree that with the increase of friendly society membership on your list there is a widening introduction, not only of the friendly society members and their dependants, but of persons outside also?

A.: Every person you contact has a wide introduction.

Q.: But I am not as much concerned with the private patient as with the friendly society one. You agree that the friendly society contact has these advantages?

A.: What advantages?

Q.: The advantages that I am speaking of, the introduction to an increasing clientele?

A.: As has any other person.

Q.: You have told us something to the effect that a great number of friendly society patients come to you in relation to minor complaints?

A.: Yes, the same as any kind of patients.

Q.: Of the numbers of friendly society patients that you have on your list, how many would you say came in respect of minor complaints—how many members?

A.: That is hard to estimate. Most of them come; it is natural that they should do so; there is no charge; they get medicine free.

Q.: Is not that probably the real reason for the frequency of the visits?

A.: Yes, the system and not the person, in the majority of cases.

Q.: I think your own figures indicate only 3·5 per person *per annum*?

A.: Yes.

Q.: That is not a very marked frequency of visits?

A.: I think the reason is because I spend a great deal of time over them in the majority of cases.

Q.: Yes, but that does not affect the number, does it? It does not add to or diminish from the number?

A.: If you see a patient thoroughly there is the less necessity to see them the next day, or the day afterwards.

Q.: Within your knowledge has there been any expression of protest against either the scope of the existing friendly society contract in Victoria or the terms of remuneration, by your or your organization?

A.: Ever since I have been through I have personally condemned it.

Q.: Do you know of any resolution being carried by your organization of protest against the existing agreement?

A.: Yes.

Q.: You do. When was such a resolution of protest carried?

A.: I suppose I am at liberty to say.

Q.: Yes, we want the fullest information. We are very anxious to get it?

A.: I think it was two years ago, and I think again more recently that the matter has been brought up. I think at the time before (I am not aware of the actual facts of the case), but I understand that during the depression it was brought up.

It was arranged that Dr. Kent Hughes should return on Thursday, October 6, for further cross-examination as to his accounts by Mr. Dovey.

The Commission adjourned at 4.30 p.m. until Thursday, October 6, 1938, at 10.30 a.m.

Thursday, October 6, 1938.

Mr. Abrahams: There are two corrections to which I desire to direct attention. There is one of those for which I am, in a measure, responsible. A statement was made on the last day of sitting, on instructions, that only 94 lodge members out of a total of 11,600 were unaccounted for. I made that statement on instructions, but I have found since that that is wrong. Four hundred and seventy-five members out of that total of 11,600 were unaccounted for. I correct the mistake, but those were my instructions at the time. There is another matter which requires correction. I have not been calling attention to errors in the notes, but this is a somewhat serious one. It occurs at page 700, about ten lines from the top [of the official transcript]. Dr. Cooper was asked by Mr. Dovey this question: "Do not you agree with what I have put to you in this respect?" He was then comparing the panel practice in London with the contract practice in Melbourne. The doctor is reported as answering: "The contract system in London is infinitely superior to the contract system in Melbourne." It would appear that either the word "Melbourne" or the word "London" has been transposed or that the word "superior" should be the word "inferior".

Mr. Dovey: Yes, I would say that there is no doubt about that being the position.

Dr. John Herbert Dorman was examined by Mr. Gain. He stated that he practised at Sunshine, an industrial area, about seven miles west of Melbourne.

He was examined as to confidential matters and then as to the manner in which he prepared his return concerning the census that had been taken. He was questioned as to the manner in which he arrived at his figures and as to the answers to the "yellow form" *questionnaire*.

Mr. Gain: You have given a figure that you expect the services to private patients to diminish by about 26%. I

think that during the year you kept an actual account of the individual patients that you attended . . . ?

A.: That is correct, individual private patients.

Q.: And you consider that, of those . . . [a figure amounting to 32·3% approximately] will come under national health insurance?

A.: That is correct.

Q.: In determining that number . . . whom did you exclude?

A.: Females over sixty and males over sixty-five, children under sixteen, and then anyone who was not an employed person with a salary of less than £7 a week or a manual labourer.

Mr. Gain asked Dr. Dorman what effect he thought national insurance would have on the dependants of people who were now private patients, but who would become insurance patients under the scheme.

Dr. Dorman: I think that once the breadwinner of the house becomes an insured person, that family on the whole will begin to develop the same sort of outlook as regards medical service. I think they will eventually reach a stage where they will want their attention cheaper. I find that in treating the dependants of a lodge patient, if they have to have an excluded service for which they have to pay, a fairly large proportion of them immediately have the reaction that they want to know if they can get it done at a public hospital, or if they can get it done, failing a public hospital, at a reduced fee; but the moment a private patient dependant comes in we tell them what is required and they leave it to us to carry out that treatment. They will tell us quite frankly what their income is and what their ability to pay is, and they will leave it to the doctor to decide where they will receive that treatment. That is what I find very prevalent. I think that once the husband becomes insured they might develop the same attitude as is developed by the majority of lodge dependants or at least by some lodge dependants.

Mr. Gain: Have you had many conversations with your lodge members as to what their position will be after they come in under national health insurance?

A.: I have not asked a lodge member directly about that, but I have had several come to the surgery and ask me what I thought about it, and my answer was that I have not started to think about it yet from that point of view. That is, if a lodge patient asks me what I think will be the outcome of it, my answer is that I have not come to any conclusions at all over it.

The Chairman: I understand that the question is directed to the capacity of these patients to pay for treatment for the dependants after they start paying the 1s. 6d. a week under the insurance scheme?

A.: What I am explaining is that I have not asked the lodge patients for this information. It has been voluntarily given to me, and in most cases they state that they do not know what is going to happen to their dependants, that they will have either to go to a public hospital or get cheaper medicine.

Q.: They ask you what you think are the prospects of somewhat cheaper medical service, is that it?

A.: Yes, they try to get a lead from me, but I will not give them a lead. I make them or let them express their own opinions and tell me what they are going to do, but they do not just know.

Q.: They do not know what they are going to do until you tell them what you are going to do in the way of charging?

A.: That is quite possible.

Mr. Gain: Has there been any discussion as to their ability to pay when that matter has been discussed?

A.: They are confident that they will not be able to pay private fees. That is the case with a lot of them that have mentioned it; but I do not say that all my lodge patients have mentioned it.

Mr. Dovey: You are speaking of lodge patients?

A.: Yes.

Dr. Dorman stated that on the whole the value of an excluded service was about equal to that of an included service, and after giving some examples concerning this point, he was asked by Mr. Gain:

Have you had any experience of people who were private patients and attended you to a certain extent and then later became lodge patients?

A.: Yes, frequently.

Q.: And were you able to form any idea as to the manner of complaint for which they would consult you whilst they were private patients and when they became lodge patients?

A.: Very definitely. As private patients I found that they would in a majority of cases be really ill; they would require a doctor's services before they came to me. I am just thinking of one family in particular at the moment, one I attended, I think, on three occasions in the first twelve months. On those three occasions there was definite sickness. Then, at the end of twelve months, they became lodge members of mine, and I have seen them since on innumerable occasions. Since then I have been treating them for such things as colds in the head and mild coughs which I am satisfied that previously they used to treat by means of a home remedy or with a bit or liquorice or something of that sort; that is, they would treat themselves. Now they come in and get a bottle of medicine from the doctor. That is a very definite case with that family.

Dr. Dorman was then cross-examined by Mr. Dovey.

Mr. Dovey: I suppose you find that your lodge patients who require services outside the contract come to you and do not go to another doctor for them?

A.: Oh, no, they come to me.

Q.: You said to the Commission, as I understand it, that the lodge patient is more prone to visit you than is the private patient? I want to ask you some questions about the number of those services that you say you have given to the patients, and I want you also to understand that in asking you these questions and in endeavouring to arrive at the manner in which you get your figures, I am not making the slightest reflection upon your honesty or *bona fides*. I feel sure that you, like everybody else who has come here, is doing his best to assist the Commission. My experience since the Commission started has been that every witness has done his best in either his calculation or his estimate, and if I have tested those estimates or calculations it has not been with any idea of reflecting upon the integrity or honesty of any witness, but has simply been an endeavour to test the accuracy of his calculations. I feel sure that my learned friends who are appearing for the British Medical Association would not have it otherwise than that those figures should be tested, because if I were to sit by and then submit hereafter to the Commission that these were mere estimates or guesses, my learned friends would say that I should have tested and probed matters when the witnesses were in the box, and given them their opportunity of making their answer then and there.

Mr. Abrahams: We court the fullest inquiry and say that here and now are the place and time to make that inquiry.

Mr. Dovey: I simply wished to show that no witness need have any fear in coming to this Commission to give evidence that any question will be asked of him which will in any way embarrass him or cause him any trouble to answer. An examination of your services to contract members shows that on your first figures you gave 6·4 services per person, and on your amended figures 5·1. Either of those figures is higher than the average, and for that reason I should like you to tell us, if you can, how you arrived at that figure [quotes the total figure]. Is it an actual count?

A.: It is an actual count.

Detailed cross-examination as to figures and the manner in which Dr. Dorman arrived at those figures followed. He was then questioned by Mr. Slater concerning the friendly society side of his practice, and later withdrew.

Dr. Robert Edgecombe Short was examined by Mr. Gain. He stated that he practised at Lilydale, about twenty-four miles east of Melbourne. He gave figures as to lodge practice for the year ending June 30, 1937.

Mr. Gain: I think that you some little time ago commenced to direct your attention to the question of the

value of excluded services per service to included services?

A.: Yes, I did.

Q.: And before you actually made any calculations you had a certain impression. What was it?

A.: I had a very definite impression that the excluded services per service were more valuable than the included services.

Q.: And then did you investigate the position?

A.: To my great surprise, I found that they worked out very much the same.

Q.: Was that before or after seeing the accountants or any of the legal advisers?

A.: I do not know that seeing the accountants affected my point of view. I worked it out and found that that was the case.

The question whether mileage was to be measured as the crow flies or upon actual distance travelled was then raised. Mr. Dovey stated that he would endeavour to ascertain upon which basis the National Insurance Commission proposed to operate, so that the matter could be dealt with later if necessary.

Dr. Short was then cross-examined by Mr. Dovey upon the figures he had given and the manner in which he had arrived at them.

Mr. Edgar Henry Ward, the financial secretary of the Victorian Branch of the British Medical Association, was examined by Mr. Abrahams. Mr. Ward gave evidence as to conversations with Mr. Gawler, the Victorian Government Statist.

Mr. Abrahams: Did you tell Mr. Gawler that your branch proposed to make a census of lodge members?

A.: Yes.

Q.: To ascertain the number of persons in a lodge membership unit, that is the dependants?

A.: Yes.

Q.: I think you told him that the object of the census would be to prove or disprove the National Health Commission's figure of 3·2 for that unit?

A.: That is so.

Q.: What did he say?

A.: The question was as to the number of persons that the census should cover. I had a discussion with Dr. Embelton, and Dr. Embelton wanted to take somewhere about thirty thousand or forty thousand. I pointed out that that would involve a lot of work and that I thought probably ten thousand would be sufficient. So I rang Mr. Gawler up and he said: "Yes, if you take a census covering ten thousand, provided it covers a fair cross-section of the whole community, it would be all right." So I asked him what he regarded as a fair cross-section, and he said: "Well, you would have to take a number of persons in the industrial areas, a number of persons in residential areas, a number of persons in provincial towns, and a number of persons throughout the country representative of pastoral and the other country interests." So then I asked him what he thought of the ten thousand, would that be a fair proportion of country members? He said he thought approximately 2,000 would give a very good result.

Q.: Was anything said about inner and outer suburbs?

A.: Yes, the industrial suburb, the inner suburb, and the outer residential suburbs.

Mr. Ward then described the manner in which the medical practitioners were selected for the purpose of the census, as to the distribution over the State and in Tasmania, and as to the choice of men likely to be accurate in the compilation of the figures.

Mr. Abrahams: And I think ultimately you selected 17 country doctors to 13 city or suburban doctors?

A.: Yes.

Q.: I think the country doctors, of course, represent a smaller number of lodge members than the city doctors?

A.: That is so.

Q.: But then the position is, of course, that there are a far greater number of lodge members in the city than in the country?

A.: That is so.

The manner in which the census had been taken was further described, documents and letters being tendered to show that there was nothing peculiar about the method, and to let the Commission see everything that had been done. Various lists and summaries were tendered as exhibits and discussed. Of one exhibit Mr. Abrahams said: This shows that of the total number of lodge members in the census, the figure in red, right at the outset of the column, 62·41 of the total are married men with dependants. That is group 1. 9·01 are widowed and single members with dependants, 16·08 are male, single men without dependants, and 12·5 are females, single people without dependants, the total making 11,332 or 100%. The 11,332 have 16,291 dependants. Of those 16,291, 94·66 belong to married men, 5·34 belong to widowers or single men, making 100%. The age classifications are shown with their various percentages. The member's age is shown in the first column, the age of the wife is shown in the next column, and the ages of the children are shown in the third column.

Further discussion ensued on the figures and documents.

Mr. Abrahams: I understand that copies will be made and that at present you have here one document prepared under your supervision showing the information extracted from those returns. You show that ten doctors whose names are given have 2,435 lodge members and that the number of attendances by those doctors on those members themselves came to 9,182, so that the attendances are 3·8 per member?

A.: That is so.

Q.: You also show that the attendances on the beneficiaries, including the members, came to 22,051?

A.: That is so.

Q.: Representing 9·1 per member?

A.: That is so; but that calculation has been done very hurriedly and there may be mistakes; for instance, there may be six-monthly periods taken instead of yearly periods. I will have those figures checked.

The Chairman: We have been told that probably there are more attendances per dependant than per member.

Mr. Dovey: Very many more.

The Chairman: Very many more in the total of attendances on dependants than in the total of attendances on members, but what is the comparison per head as regards attendances on dependants and members?

Mr. Abrahams: I should say about the same.

The Chairman: I should have thought it was rather more. Apparently from the figures which you have there, there are slightly more attendances on members than on dependants per head.

Mr. Dovey: In many cases there are two to three times as many attendances on the wife as on the husband.

Dr. Eric Mendel Ettelson, of East St. Kilda, was examined by Mr. Gain. He gave evidence as to his lodge practice, producing figures for a month, which were tendered as a confidential exhibit. He also gave figures as to his private practice. Dr. Ettelson then gave evidence as to his experience as *locum tenens* in various panel practices in England. He was asked by Mr. Gain to contrast the service in a certain panel practice in England with the service in contract practice in Australia.

Dr. Ettelson: I would say that it was not nearly as good. There was one thing I noticed in regard to the panel practice, and that was that they had no what I would call surgical sense at all. They did not have any proper surgery. This man, for example, was a very conscientious gentleman, and yet he was not capable of doing the slightest thing in the way of surgical procedure with any skill whatsoever. He could not put a stitch in decently. He had no proper idea of cauterizing, and, as a consequence, I found that anybody who had a surgical condition did not get good treatment from him. He endeavoured to give conscientious service to his panel patients and to his private patients alike. If a man came in with a cough, he got him to take his shirt off and he examined him. In the ordinary panel practice that would be almost unheard of, but, if he had to do anything that required a surgical dressing, the job was not well done.

You cannot have a man who never does any surgery at all, who never puts his hand to surgery, because he loses all skill in that direction. When I first went there, I attended those patients for him, and, after a while, I asked him whether he would take them over because I was so worried about the conditions under which they were living. They appeared, to be satisfactory to him, but they were not satisfactory to me. I had a similar experience in regard to lack of surgical procedure . . . the doctor for whom I acted as *locum tenens* was a particularly decent fellow and a very nice gentleman . . . He was running the V.D. clinic there, and the first night I went there to run the clinic for him, I had to give an injection for him to a man, and the assistant went straight to a cupboard and handed me a syringe and a needle. I said to him: "This has not been boiled." He said: "We never boil them."

Q.: That is to say they never sterilize them?

A.: Quite so.

Q.: Was this to a private patient?

A.: No, to a public patient, but nevertheless he would not have done any more in regard to a private patient.

Q.: Was this to a panel patient?

A.: I am not suggesting that the treatment given to panel patients was worse than the treatment given to private patients.

Q.: You are now dealing with the common practice?

A.: I am saying that the practitioner is conducting his practice in the same way for the panel as for the private patients.

Mr. Dovey: Your remarks in respect of this practice apply equally to the private patients as they do to the panel patients?

A.: Yes, it is both the same. There was one private patient who had to have a catheter. He was an old man and he had to have a catheter every day. When he came along he just picked up the catheter off the sideboard and just passed it.

Q.: With no sterilization?

A.: No, none at all. In regard to minor surgery, none was done. I spent a great deal of my time in Cornwall doing minor operations, which had not been done for years. I liked doing them, because it gave me something to do and gave me some interest in the work. The same thing happened when I was . . . in an industrial area in London.

Q.: Was that a mixed practice also?

A.: Yes. There was a detached surgery there. I went there one day for him and there was a patient came in who required a couple of stitches to be put into a small cut. I forget now where it was, but I hunted around the surgery and I found a very old rusty needle and a rusty old pair of forceps. I also found an electric kettle, but I could not find any stitching material. I then went around the corner to one of Woolworth's shops, and I bought a roll of something suitable for stitching. I then boiled it in the electric kettle and inserted a couple of stitches. I came away feeling rather pleased at myself for showing a certain amount of initiative. I met the doctor's son, who was quite an intelligent fellow, and I said to him: "I had to put a couple of stitches in a small cut, but I could not find anything but an old needle, an old pair of forceps and an electric kettle. I had to boil the stitching I got in the electric kettle. He said to me: "Father never bothers to do that."

Q.: Did you notice anything about the amount of attention required by the panel patients in England as distinct from the lodge patients in this country?

A.: Yes, it was my opinion again that the panel patients require a very considerable amount more treatment for minor ailments than they do here. There was also a lot of time spent in giving prescriptions for things which you might almost call household supplies. For instance, patients would come in and ask for a prescription for three packets of Epsom salts, or a bottle of vaseline, or half a pound of cotton-wool.

Mr. Dovey: And would you give it to them?

A.: Yes. As a matter of fact you would have to give it to them, because, under a system such as they have there the thing that impressed me was the constant fear that the decent men had, and there are decent men in panel practice. That is a thing I want to emphasize. I do not want to run down the British medical profession . . .

Dr. Ettelson gave further evidence as to conditions in England both in panel practice and in hospitals. He was then cross-examined by Mr. Dovey upon his evidence as to conditions in England, and by Mr. Slater upon his experience in lodge practice in Victoria.

Dr. M. O. Kent Hughes, of Moonee Ponds, was recalled and cross-examined by Mr. Dovey as to the manner in which the returns for the census were secured by the witness. The cross-examination was not completed when the Commission adjourned.

The Commission adjourned at 4.30 p.m. until Friday, October 7, 1938, at 10.30 a.m.

Friday, October 7, 1938.

Dr. M. O. Kent Hughes was further cross-examined by Mr. Dovey. He was questioned concerning the manner in which his return for the purpose of the census had been compiled.

Mr. Dovey: It seems to me to be important, Your Honour, to ascertain, in respect of as many as possible of those doctors who furnished the information, to what extent the information is given, as it were, first hand; that is to say, direct from the lodge member concerned, and how much is given from the information and belief of the medical practitioner.

The Chairman: If it turns out that a considerable portion of the information resulting from the census is based upon the information and belief of the practitioner, and not upon the direct statement of the lodge member or of an official lodge, it will affect our minds as to how much, if any, of the result should be discounted. In connexion with returns of this kind, which are really a statement of an estimated average, and not an absolute result at all, one always has to pay regard to the circumstances attending the gathering of the information, so as to see whether the result should not be discounted to some extent. One has to be cautious in accepting results of this kind, so that you are quite right in finding out, as far as you can, how much of the information gathered is simply based upon a doctor's estimate, as distinct from direct information from those who know.

Mr. Dovey: That was what I had in mind.

The Witness: May I submit these cards. They are twelve cards taken at random, stating the reason why the circulars were not sent out. They were people with whom I had personal contact all the year, and you will see that I have seen most of them and know them. The circulars could easily have been sent out.

The Chairman: A statement of that kind by a medical man who knows the family and so on is, in my opinion, of equal value to an actual statement by the lodge member himself. One cannot make any difference in the weight to be given to it; but where a statement of a medical man is simply based upon a kind of general impression, it is not of much value?

A.: These are not general impressions. They have been gone into. The others were either contacted by circular or by personal touch, and great care was taken.

Dr. Kent Hughes was then examined as to the circumstances in which the information contained on certain specific cards was obtained.

Mr. E. H. Ward was recalled and further examined by Mr. Abrahams. Mr. Ward produced a summary showing the result of the analysis of the figures for the attendances on members and dependants which had been supplied by certain doctors.

Mr. Abrahams: Before we deal with the adjustment sheet, is this the final total for the 10 doctors—2,435 members, representing 6,168 persons?

A.: That is so.

Q.: Attendances on the member 9,937, which is an average of 4·08; total attendances on the member and dependants, 24,917, representing 4·03 per beneficiary?

A.: Yes.

Mr. Ward was further examined as to the summary which he had produced. He then withdrew.

Dr. George Rankin Bearham was examined by Mr. Gain. He stated that he practised at Thornbury, an industrial artisan suburb, about five miles from Melbourne, and that he was one of the medical practitioners concerned in the taking of the census. He estimated the loss of private practice at the figure of 37%, which was determined on a count of heads of the private patients who visited him.

Dr. Bearham, when cross-examined by Mr. Dovey, gave a description of the manner in which he recorded his attendances and kept his records. He was then examined as to the census-taking and the methods adopted by him in making his returns for this purpose.

Questioned by Mr. Slater concerning his lodge practice, Dr. Bearham stated that his lodge panel had been increasing but that he had not objected to this increase, although he did not feel satisfied with the remuneration.

Mr. Slater: Would you be surprised to know that no representation has ever come from the British Medical Association to the Friendly Societies' Association until March of this year seeking any variation of the Wasley award?

A.: That has nothing to do with me.

Q.: Would you be surprised to know that that is the position—that there have been no representations?

A.: And no discussions before then?

Q.: Yes, would you be surprised to know that there have been no such discussions or representations or moves on the part of the British Medical Association for greater remuneration?

A.: And that there have been no meetings between the British Medical Association and the leaders of the lodges over it?

Q.: Yes, you can put it in those words too?

A.: I would be surprised to know that.

After further examination as to the returns which he had made and as to his practice generally, Dr. Bearham withdrew.

The further sittings of the Commission were adjourned until Tuesday, October 11, 1938, at 10.30 a.m., at the Coroner's Court, Victoria Square, Adelaide.

Correspondence.

THE TREATMENT OF CANCER OF THE CERVIX UTERI.

Sir: *Re* Drs. H. Ham and R. A. Gardner's letters printed in your correspondence column.

We have no intention of entering into a controversy regarding the relative merits of different forms of treatment for cancer of the *cervix uteri*, and our statistics were not given in the spirit of competition, but to fulfil a promise given to Professor Sandes, the director of cancer treatment, some five years ago. If those responsible for other forms of treatment had carried out their part of that bargain we would have had local statistics that would have been comparable. We await these results.

The following extract by Professor Fernand Bezançon, *Formulaire Astier*, 1937, on the subject of uterine cancer, represents almost exactly our own views, and is of importance as representing the feeling on the subject in France, the home of radiotherapeutics:

Cancer du col.—Les indications respectives du traitement sanglant (hystérectomie abdominale totale élargie ou Wertheim) et de la curiethérapie ou de la radiothérapie profonde font l'objet de discussions et l'accord n'est pas entièrement réalisé. Cependant, il semble que pour les cas tout au début, cas où l'opération doit être facile, le traitement de choix soit le traitement sanglant. Doit-on faire suivre le Wertheim de séances de radiumthérapie? Sur ce point, les avis sont partagés mais le plus grand nombre de chirurgiens penchant pour l'affirmative. Pour les cas un peu avancés, ceux pour lesquels on a des doutes sur la souplesse du paramètre, le traitement de choix est le traitement par le radium suivi de séances de radiothérapie profonde. Mais ce traitement n'est pas sans présenter des dangers graves s'il est appliqué très complètement; d'autre part, il existe des néoplasmes radio-sensibles et d'autres radio-résistants. Doit-on faire suivre le traitement par le radium de l'extirpation chirurgicale, rendue ainsi possible automatiquement? Le plus grand nombre de chirurgiens s'abstiennent en raison des difficultés. Enfin, soit primitivement, soit pour poursuivre des récidives, on a proposé d'aller placer directement les tubes de radium dans les ligaments larges par laparotomie. La radiothérapie profonde n'a pas donné tout ce qu'elle promettait. Pour les cancers très avancés, on peut se trouver bien de quelques courtes applications de radium sur les bourgeons, dans le but de diminuer les hémorrhagies. L'air chaud (à 300° environ) calme, désinfecte, désodorise et rend aussi de grands services quand on ne peut plus espérer de traitement curatif.

Cancer du corps.—Hystérectomie totale à la condition que le diagnostic soit précoce.

Yours, etc.,

Herbert H. Schlink.

Clement L. Chapman.

"Craignish",
185, Macquarie Street,
Sydney.
September 27, 1938.

FRIENDLY SOCIETY LODGE PRACTICE.

Sir: In the course of conversation with the secretary of one of the local branches of a friendly society, he told me that he had found recently that a prospective member of his lodge is in receipt of an income exceeding £260 *per annum*, and informed him that he would be ineligible for medical benefits. The prospective member then did not proceed to membership. This secretary mentioned this matter at a meeting of friendly society delegates at a district centre, and was informed by others present that it was not his business to see that persons in receipt of incomes above the limit were not placed on a doctor's list—that other secretaries are not as particular as he. My friend contended that as this limit is fixed by the common form of agreement, and as the member states on a form completed before initiation to the lodge whether he has been in receipt of more than £5 per week for the past twelve months, it is the duty of the secretary to inform him that he is ineligible for medical benefits if his earnings exceed this amount.

Clause 2 of the common form of agreement states that "The Medical Officer shall . . . upon payment to him of a fee of two shillings and sixpence examine every person seeking to become entitled to any of the benefits of the said Lodge, who shall bring to him a statement in writing signed by the Secretary . . . notifying that such person, if passed by the Medical Officer, is entitled to such benefits . . . according to the terms of this Agreement, . . ." It appears that some lodge secretaries are not conversant with this clause, and it is of great importance to the profession that they should be reminded of its existence and of the fact that it throws the onus upon them to safeguard their medical officers in the matter of the income limit. I suggest that the reminder should come from the Council of the New South Wales Branch of the British Medical Association.

The vexed question of lodge members who pass the second income limit of £364 *per annum*, and yet continue to expect and accept medical benefits, is one which should be considered seriously at this time. Most, if not all, medical men doing lodge work find it expedient to overlook this imposition; but if as a profession we are foolish enough to accept any form of government-controlled health insurance scheme, then it will be very galling to have to continue to treat as friendly society members a fairly large number of men and their families who should be amongst our few remaining private patients.

Yours, etc.,

R. Errol Maffey.

"Kanimbla",
Singleton,
New South Wales.
September 29, 1938.

THE PSYCHOLOGICAL APPROACH.

Sir: Dr. R. N. Beazley, like many others, is anxious to find an actual working method which has given good results in psychological disturbance.

There are at least four well-known methods in general use, depending upon the suitability of the patient, the predilection of the practitioner and his criterion of cure. They are suggestion, hypnosis (personal or narcotic), superficial mental analysis and true Freudian psychoanalysis.

The first essential of any successful treatment is recognition of the psychogenic factor, and the most thorough method of therapy, in suitable cases, is psychoanalysis. This desideratum is often unattainable; but it is surprising how much relief may be achieved by applying psychoanalytic knowledge to the apparently simpler cases which can attend for a couple of months only. But let no one delude himself into thinking that this is a high standard of cure.

Yours, etc.,

E. Haslett Frazer.

231, Macquarie Street,
Sydney,
October 1, 1938.

Sir: Perhaps there is no branch of medicine of which it may be said more aptly that "of the making of books there is no end and much learning is a weariness". Only after a considerable amount of experience is it possible to evaluate and then, perhaps, apply some of the diverse and even contradictory methods advocated by the different schools of psychological practice.

Unless the student of the "Psychological Approach" is prepared to devote some time to acquiring clinical experience, mere reading will only make the confusion in his mind more confounded. Dr. Beazley refers to the fluency of some writers on the psychoneuroses. Unfortunately much of the literature on the psychoneuroses consists of attractively written biographical sketches intended to illustrate some mechanism or other, the treatment and subsequent fate of the patient being left without comment. A critical survey of psychological theories and methods, with special reference to their practical value, is long overdue.

Finally, may I call Dr. Beazley's attention to the short course in psychiatry for general practitioners which the Post-Graduate Committee proposes to hold during March, 1939.

Yours, etc.,

W. S. Dawson.

Sydney,
October 5, 1938.

Proceedings of the Australian Medical Boards.

VICTORIA.

A meeting of the Medical Board of Victoria was held on September 7, 1938. The undermentioned was registered as a medical practitioner pursuant to the provisions of the *Medical Act*, 1928, of Victoria:

Maclurkin, Alfred Robert, M.B., Ch.B., 1900 (Glasgow), D.P.H., 1904 (Edinburgh), D.T.M., 1904 (Liverpool).

The names of the following deceased practitioners were removed from the register: Warren James Fearnsley, Carl Vivian Stephens.

Ernst Cohn produced a deed poll evidencing the change of his name to Ernest Colin, and applied for alteration of the entry in the register accordingly. The application was granted.

The question of the eligibility for registration of applicants who have completed a course of less than five years' study in Italy was referred to the Crown Solicitor for advice.

It was decided to obtain the advice of the Crown Solicitor as to whether evidence obtained concerning the alleged association of a registered medical practitioner with an unregistered practitioner was sufficient to justify the institution of proceedings.

An explanation was received from a medical practitioner in relation to the advertising matter issued in support of his candidature at the recent municipal elections, and it was decided that no good purpose would be served by taking further action.

NEW SOUTH WALES.

The undermentioned have been registered, pursuant to the provisions of the *Medical Act*, 1912 *and* 1915, of New South Wales, as duly qualified medical practitioners:

Campbell, Margaret Eileen, M.B., Ch.B., 1930 (Glasgow), Guyra.

Osmond, Henry Thomas, M.B., Ch.B., 1937 (Edinburgh), c.o. Perpetual Trustee Company, Hunter Street, Sydney.

Smith, Thomas Malcolm, M.B., Ch.B., 1930 (Univ. New Zealand), c.o. Watson Victor Limited, Bligh Street, Sydney.

Books Received.

MODERN MEDICAL MONOGRAPHS: MODERN METHODS OF FEEDING IN INFANCY AND CHILDHOOD, by D. Paterson, B.A., M.D., F.R.C.P., and J. F. Smith, F.R.C.P., edited by H. Maclean, M.D., D.Sc., F.R.C.P.; Sixth Edition; 1938. London: Constable and Company Limited. Demy 8vo, pp. 230, with illustrations. Price: 7s. 6d. net.

LEAGUE OF NATIONS PUBLICATIONS. ANNUAL REPORT ON THE RESULTS OF RADIOTHERAPY IN CANCER OF THE UTERINE CERVIX. SECOND VOLUME: STATEMENTS OF RESULTS OBTAINED IN 1931 AND PREVIOUS YEARS (COLLATED IN 1937), edited by J. Heyman, M.D.; 1938. Geneva: League of Nations Publications Department; Australia: H. A. Goddard. Medium 8vo, pp. 94. Price 1s. 6d. net.

THE WHEEL OF HEALTH, by G. T. Wrench, M.D.; 1938. London: The C. W. Daniel Company Limited. Demy 8vo, pp. 146. Price: 6s. net.

MALARIA IN THE NETHERLANDS, by N. H. Swellengrebel and A. de Buck; 1938. Amsterdam: Scheltema and Holkema Limited. Demy 8vo, pp. 275, with illustrations. Price: D.Fl. 5.

THE EVOLUTION OF CHRONIC RHEUMATISM. WITH TREATMENT TO CORRESPOND: THE PREVENTIVE CLINIC AS A FIRST LINE OF DEFENCE, by R. F. Fox, M.D., F.R.C.P.; 1938. London: H. K. Lewis and Company Limited. Royal 8vo, pp. 28. Price: 2s. 6d. net.

MEDICINE IN ITS CHEMICAL ASPECTS. REPORTS FROM THE MEDICO-CHEMICAL RESEARCH LABORATORIES OF THE I. G. FARBENINDUSTRIE AKTIENGESELLSCHAFT; Volume III; 1938. Leverkusen: Bayer Pharma Limited. Medium 8vo, pp. 416.

Diary for the Month.

OCT. 18.—New South Wales Branch, B.M.A.: Ethics Committee.
OCT. 19.—Western Australian Branch, B.M.A.: Branch.
OCT. 20.—New South Wales Branch, B.M.A.: Clinical Meeting.
OCT. 25.—New South Wales Branch, B.M.A.: Medical Politics Committee.
OCT. 25.—Queensland Branch, B.M.A.: Council.
OCT. 26.—Victorian Branch, B.M.A.: Council.
OCT. 27.—South Australian Branch, B.M.A.: Branch.
OCT. 27.—New South Wales Branch, B.M.A.: Branch.
NOV. 1.—New South Wales Branch, B.M.A.: Organization and Science Committee.
NOV. 2.—Western Australian Branch, B.M.A.: Council.
NOV. 3.—South Australian Branch, B.M.A.: Council.
NOV. 4.—Queensland Branch, B.M.A.: Branch.
NOV. 8.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
NOV. 9.—Victorian Branch, B.M.A.: Branch.
NOV. 11.—Queensland Branch, B.M.A.: Council.
NOV. 15.—New South Wales Branch, B.M.A.: Ethics Committee.
NOV. 16.—Western Australian Branch, B.M.A.: Branch.
NOV. 17.—New South Wales Branch, B.M.A.: Clinical Meeting.

Medical Appointments.

Dr. N. S. C. Mulhearn has been appointed Government Medical Officer at South Grafton, New South Wales.

* * *

Dr. V. H. Leigh-Barlow has been appointed Government Medical Officer at Gresford, New South Wales.

* * *

Dr. H. T. Illingworth has been appointed Medical Officer of Health to the Local Board of Health at Youanmi, Western Australia.

* * *

Dr. C. H. Prouse has been appointed, pursuant to the provisions of the *Workers' Compensation Acts*, a Certifying Medical Practitioner and Medical Referee at Werribee, Victoria.

* * *

Dr. N. S. Williams has been appointed Medical Officer of Health to the Balingup Road Board, Western Australia, in accordance with the provisions of *The Health Act*, 1911-1937.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xviii to xx.

CHILDREN'S HOSPITAL (INCORPORATED), PERTH, WESTERN AUSTRALIA: Junior Resident Medical Officers.
FREMANTLE HOSPITAL, FREMANTLE, WESTERN AUSTRALIA: Junior Resident Medical Officer.
INFECTIOUS DISEASES HOSPITAL BOARD, FAIRFIELD, VICTORIA: Junior Resident Medical Officer.
RENWICK HOSPITAL FOR INFANTS, SUMMER HILL, NEW SOUTH WALES: Resident Medical Officer.
STATE PUBLIC SERVICE, QUEENSLAND: Assistant Medical Superintendent.
THE WOMEN'S HOSPITAL, CROWN STREET, SYDNEY, NEW SOUTH WALES: Honorary Officers.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 178, North Terrace, Adelaide.	All Lodge appointments in South Australia. All contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.

Editorial Notices.

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